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THE NEUROLOGIC DILEMMA*

SIDNEY I. SCHWAB, M.D.

ST. LOUIS

The opening chapter of William James' "Pragmatism" is called "The Present Dilemma in Philosophy." In place of philosophy read neurology, and the title of this president's address is revealed. The minor differences between James' chapter and mine will be found in the fact that I propose to devote just twenty minutes to the presentation of the dilemma in neurology. The major difference I shall refrain from mentioning, because I want to feel for a few moments, at least, worthy of the honor you have conferred on me.

It is far from my purpose to suggest a pragmatic solution, however, but rather do I want to certify to the existence of a dilemma and then to ask you to note that its partial solution can be found in the present program which my paper so feebly initiates.

Dilemmas exist only when attention is directed to them or when something happens which disturbs the orderly working of events and an effort is made to understand why things are not as they once were. I began to be dimly aware that there was something the matter with neurology when it was brought to my notice that in the neuropsychiatric service of the American Expeditionary Forces there were only a few neurologists, and that about 90 per cent. of the service was made up of asylum or institutional men. The war neuroses were essentially problems of neurology, and neurologists as a whole had very little to do with the attempt at solution. Whatever the explanation turned out to be is now of little importance, but this extraordinary lack of neurologic participation in one of the most significant neurologic opportunities of this generation led irresistibly to the conclusion that there were counteracting and antagonistic tendencies in neurology. It is out of these that the dilemma I am now going to talk about originates. It is my purpose then to touch briefly on the present state of neurology, to indicate what neurology seems to me to be, and to dissolve the dilemma by stating the things that make it up. As this address is not to be open to discussion, now at any rate, I shall find it comparatively

*President's address read at the annual meeting of the American Neurological Association, Atlantic City, June, 1921.

easy to state such thoughts on the subject as I happen to have with a degree of boldness and definiteness not usually characteristic of presidents' addresses in our time and in our country.

A BROADER CONCEPTION OF NEUROLOGY

Neurology just now presents a most curious and interesting anomaly, owing to the fact that the personal and clinical treasures of the neurologist are really no longer his. He is permitted to have only a far distant and often impersonal interest in them. One by one the old and classic standbys, the dead ends and fruitless scars of ancient processes are dealt with by those to whom a neurologic training and type of thought have been denied. Syphilis of the nervous system, tabetic and paretic reactions are all now well within the territory preempted by surgeons, dermatologists and internists. They make the diagnosis, plan and carry out treatment, often with skill and intelligence it must be admitted, without the least idea that they are treading upon ground discovered and cultivated by a preceding generation of intrepid neurologic thinkers. I mention this merely to indicate the steadily narrowing fields of activity left to those whose neurologic horizon is limited by the traditions of the past; traditions which are crumbling under the attacks of our more venturesome colleagues. I shall leave to your imagination the rest of this story. Rare spinal cord lesions, the curious and unusual types of disease, familial deviations, congenital anomalies upon whose unending diagnostic and therapeutic difficulties the neurologic intelligence wages a hopeless struggle, are the residuals left after the piratical sifting is finished. These may well be called dead ends in contrast to the live and vivid realities which a broad conception of neurology should encourage us to put our teeth into. I am leading perhaps somewhat deviously to the first point which I want to make, and that is that neurology must be redefined and we must be courageous enough to follow where this redefinition leads. Neurology is concerned with the totality of the problems presented by the nervous system in health or disease and any condition influenced by its disturbed function. Consciousness is perhaps the chiefest of these functions, and to its expression, whether normal or abnormal in the accepted meaning of that term, neurology and neurologists must be actively concerned. This points directly to that group of defects which neurologists have hitherto been only half-heartedly interested in—the psychoses. I am not referring to the legal and custodial phases, but to the direct participation in the study, care of, and direction of the insanities as they are met with in the daily experience of all of us. I desire to accentuate the conclusion that there is no psychiatric point of view, that there is no such thing as a psychiatric method of thinking or approach which logically can be divorced from the neurologic one.

It is the latter that contain the vital elements of scientific advancement. When the problems of consciousness are neurologically approached then progress will begin.

THE NEUROLOGIC POINT OF VIEW

What is meant, then, by the neurologic point of view? It is briefly this: What knowledge there is of disease of the peripheral nerve, spinal cord, and brain based on lesional defects was arrived at through anatomic and physiologic methods of thinking applied to conditions found in the nervous system. A mass of definite, tangible, and illuminating information was acquired, which, crystallized by observation, experience and thought, furnished the data of neurology. This attracted the attention and awakened the interest of neurologists from the period of S. Weir Mitchell and Charcot, roughly speaking, to the time of the war. The methods that grew out of their activity and out of the work of many other men of their stamp stood the critical test and were productive in establishing what is now in many respects a well correlated and reasonably well known field of knowledge. Such methods should be followed, modified in ways that can only now be surmised, when the problems are less tangible, less material, and, unfortunately, more complex.

The abnormalities of consciousness, expressed clinically in psychoses, become the objectives of neurologic endeavor as the logical development of the neurologic method reaches out to its ultimate possibilities. I am of course speaking of methods of thought, not technic of examination. The certainty of these conclusions appears to rest upon the fact that there is no fundamental difference in intellectual processes which can be said to be dependent on differences which are innate in the object of inquiry. The human mind in its function of thinking is not modified and does not undergo a change because the subject under scrutiny possesses one quality or another. The assumption that this is necessary causes the dilemma which has so long kept the neurologic method focused so largely upon purely anatomic problems. There is no fundamental difference in scientific method in the attempt to work out the factors that go to the making of a diagnosis of tabes, for example, and that which is concerned in manic depressive psychosis, dementia praecox, or a neurosis. If the method differs, and when it departs from the physiologic, anatomic, or chemical type of reasoning, then there is a defect in the thought process, and defective diagnostic and other conclusions are the result.

SENSORY DATA

In using the terms physiologic, anatomic, and chemical, I should like to include also such psychologic methods as are based on sensory

data. This, however, by no means necessitates limiting in any way the sources out of which the essential data comes; but it is necessary, I think, to measure and weigh such data by the critical and objective qualities of neurologic thought, in opposition to the mystic or introspective methods which are deemed so essential when neurology is thought of as a domain limited and bounded by the peripheral nervous system on the one hand and tumors of the brain on the other. I fancy that the neurologist's objection to a great deal of the freudian conception of the neuroses is not due to the conclusion arrived at but to the loose methods of thinking indulged in. The assumption that a thing is correct because the expected is proved is much less convincing neurologically than if the unexpected sometimes happens. The sexual etiology of the neuroses does not strike terror to the neurogolic heart, but it is the prophecy and expectation that nothing else can be the explanation that is annoying and irritating.

There is here detected a faulty method of thinking which arises on account of the insistent and initiatively controlled trend which is wholly at variance with the free play of the trained intelligence upon a difficult and intricate problem. Psychanalysis as a method of investigation is possibly adequate, but it ceases to be so the minute it departs, in any important way, from the influence exerted by the deductive procedure developed in the building up of a neurologic method. In the investigation of the neuroses the same method of inquiry must be followed. These phenomena of an altered function of the nervous system are capable of investigation by the same methods of thought that are used when the object of inquiry happens to be a localized lesion of the spinal cord. The control exercised by the postmortem study is unfortunately lacking in the former of these, but in the clinical problem no difference in the method of approach is necessary. Causation, mechanism, and clinical end results are there just the same, even if the final test of the microscope is at present lacking. After all, the method is the main concern, and the scrutiny of final conclusions is to be governed accordingly. In the physiology of the nervous system is to be found the key to the neuroses, and from it will eventually arise the laws governing the grouping, distribution, and quality of the sensory objectivation of hysteria, just as definitely set as those produced by a tabetic lesion of the posterior root and column.

Please do not think I am advocating a physiologic parallelism. I have no definite ideas on this subject whatsoever. I am merely calling attention to methods of thought and ways of thinking.

It is obvious, therefore, that the field of neurology must of necessity embrace that aspect of deviation of the nervous system produced by anatomically demonstrated changes in its structure, that disturbance

in its correct function that is called a neurosis, and that failure in adaptation which is termed a psychosis. This, I believe, is the field of neurology. This wide embrace is necessitated by the fact that there is no definite dividing line between them, and that the best and most fruitful method of approach is that which has had the best success in the understanding of the more obvious and more tangible problems, those that belong to the anatomic group.

THE NEUROLOGIC DILEMMA

I have tried to lay bare the elements out of which the dilemma in neurology has arisen. The dilemma, then, is this: The neurologist is caught in the meshes of contradictory tendencies in the subject by which its limits in one direction are steadily receding and those in another are extending beyond his visible horizon. He lacks the courage to wander into territory preempted by what appears to be a defective method because he lacks the conviction and faith in the correct method. He is too readily impressed by the conventional and traditional belief that some mysterious quality of mind is needed in order to venture into a land which by virtue of his training and experience he is best fitted to explore. I am convinced that the psychiatric phase of neurology, if such a paradoxical expression be permitted, is a definite part of any neurologist's activity and that the old separation of the subject should be given up. This does not by any means imply that there need be any limitations of specialists' activities in any direction. The neurologist may well choose one or another field or one or another group in which he is particularly interested. The whole, however, should be neurologically directed and infiltrated by the best neurologic thought. The solution of this dilemma must come about by keeping in mind very steadily this fact: neurology suffers when it separates itself too far from the problems of medicine as a whole. Psychiatry has, I believe, been artificially separated therefrom, and by its separation and segregation progress has been made unusually and uselessly difficult. The closer both get to the problems of medicine, the greater the advance will be, and if both do this they will automatically merge into the common subject to which they belong, which is disease of the nervous system. The place for neurology in the conception that I have in mind is a general hospital, where it should find its physical home. From this its material comes, and out of this combination its progress will develop. In a practical way, therefore, the university neurologic clinic will arise, as it is beginning to show itself in a few places scattered throughout this country. The totality of the problems of the nervous system will become, then, the object of inquiry, and the subject of this inquiry will be found in the persons whose nervous systems are diseased.

If it once becomes possible to view the nervous system as a whole, not only in its normal condition, but also when it becomes the subject of disturbing factors which produce what is called disease, the dilemma that confronts the neurologist will surely disappear, because it is the lack of a convincing method of approach that gives the dilemma its vitality, not the innate difficulties of the position itself. The impasse of a dilemma is usually brought about by the contradiction of tendencies, a pull in diverse directions. A stabilizing of effort or a neuteralization of energy results, and the thing that can be solved remains unsolved. In an attempt to rationalize this lack of progress the difficulties are assigned to a place where they do not belong, and a terminological defense system arises, which perversely enough becomes the dilemma itself. Against the false screen the efforts at solution are directed, and no wonder they so often fail. It is a fortunate thing that neurology has been able to build up for itself a scientific method that has all the characteristics of workability and adaptation.

Contrasted to this is a method of thought that derives from a prescientific era, in which what was inside of one's mind rather than the data outside was made use of. I make use of this rather crude comparison to point out the striking contrast between what I conceive to be the opposing thought tendencies in the two branches of nervous diseases to which the horns of our dilemma are directed. That this divergence of thinking exists can be easily proved by noting the mental behavior of a medical student in the presence of a case of tabes and a case of dementia praecox. The ease with which the former is examined and the awkwardness and embarrassment in the presence of the latter are striking indications of what I mean. As soon as a case of paresis is presented to him and it is indicated that the process here is identical with that of tabes, the contrast of thinking almost immediately disappears. The inclination to cling to the objectivation of symptoms and to feel a certain sense of comfort in the thing seen or felt is an almost pathetic reminder of the artificially produced breakdown of the neurologic method of thinking in the face of symptoms arising from disturbed mental functions. This is where I have tried to put my finger on the sore place out of which the dilemma arises.

THE TECHNIC OF MENTAL TESTS

It is the unwarranted assumption that a different kind of thinking is necessary in the one problem and its sudden realization that produces a blocking of smooth effort in the observation and noting of data and the necessary conclusion from them. The technic of a student in a case of dementia preacox in its neurologic, medical, and psychical phases is an interesting commentary on the clash of methods. The contrast

to my mind should not exist at all. The use of material methods of observation as contrasted with those to which the term psychic is given is certainly striking. When the physical, neurologic, and chemical tests are over the examiner pauses as though to say, "Now I am going to examine this patient's brain or mind." Immediately there is a change in his attitude, a change which I believe is detrimental to such knowledge as he is able to obtain. What I am particularly interested in noting, however, is that with this change of attitude really goes no important fundamental change in method, because when it comes to the point of making a mental test or mental investigation or mental analysis, use is made and can only be made of the same channels of study that were used in the examining of the reflexes, or sensory tests, or what not.

After all is swept aside and all the hypertechnical phrases are done away with, there remains the method of question and answer. The instruments by which this is accomplished is by speech and by hearing. Everything else is adventitious, and what goes on in the patient's mind and comes into the examiner's mind does so through the sensory channel alone. It is the receptivity of one mind in the face of the attempted expression of the content in another's that satisfies all of the legitimate demands of the examination into the psychic field. Technic consists, if there is any such thing as technic in this sense, in the use of analysis, judgment, memory, the weighing of evidence, the use of past experience, the use of observation of facial expression, the reaction quality of emotion, etc., and if one examines this mass of data and the other mass of data he is not aware that there is any distinction of any importance whatsoever. The dilemma here consists in not admitting frankly and honestly that the difference in method is merely a difference in terminology; and, if it were not for the fact that terminological emphasis is placed on one method to the exclusion of the other, the neurologist's thought processes in one would be exactly the same as in the other. It is when the neurologist is influenced by the dilemma that arises in such an instance that the method itself begins to suffer, and unconsciously there is substituted something which is called psychiatric, and with which he feels unfamiliar because he instinctively knows that it is wrong. What I mean to imply is simply this: So far as psychiatric method is effective and productive it shares with neurologic methods a correct system of thought; when it departs it is manifestly defective. The dilemma here arises out of the impulse to dodge the hard issues of the neurologic pathway and to follow that part of the psychiatric method which falsely promises a way out. The curious thing is that it is this false element to which the term psychiatric is found most often applied.

CONCLUSION

The dilemma as it shows itself to me is made up, as most dilemmas are, of a series of false ideas, of illusionary elements, magnified into a semblance of reality by a terminology that finds a certain amount of attractiveness in its tautological nuances. Its chief incentive toward escape is based upon the false notion that because a thing is expected, all things are wrong that do not lead to that expectation.

The dilemma in neurology disappears as soon as it is faced or as soon as it is understood. The way out lies in the utilization of neurologic methods of thinking. The neurologist's opportunity is the nervous system, and no problem there is less than his right of possession nor more than his capacity, modestly and courageously tempered, may not hope to solve. Thus the dilemma vanishes and if, in attempting to dissolve it, I have not been able to state my thoughts with the grace and felicity of the great philosopher whose title I have dared to borrow, yet I hope I have been touched a little by his honesty of purpose and his supreme intellectual courage.

Humboldt Building.

INTRACRANIAL TELANGIECTASIS

REPORT OF TWO CASES *

E. M. HAMMES, M.D.
Assistant Professor of Neurology and Psychiatry, Medical School, University of Minnesota
ST. PAUL

The two following cases of probable cerebral telangiectasis seem to be of sufficient interest to warrant report.

REPORT OF CASES

CASE 1.—*History.*—A merchant, aged 21 years, was referred by Dr. Ransom of Hancock, Minn., July 14, 1920. The patient's family history was negative except that his father died of diabetes. When 7 years old, he was thrown from a horse and struck on his head, but he remembered no details of the accident; appendectomy was performed on him when he was 14; he denied venereal disease. When 8 years old, while convalescing from an attack of mumps, he had a severe convulsion and remained unconscious for fourteen hours. He recovered and was well for three weeks, when he had another mild attack. Since that time the number of attacks have varied from three in one day to one in six months. He described a typical attack as follows: It begins with a sensation of electricity in the left hand, after which he feels as if the hand were off. Within a few minutes, he notices a jerking of the fingers of the left hand, gradually involving the muscles of the entire left arm. When it reaches the shoulder, he becomes semiconscious and feels as if he were floating in space. During this semiconscious period, he has excruciating pain in the left arm. Then he loses consciousness for a period of from three minutes to two hours. When he recovers consciousness he is confused and talks irrationally for a short time and his left arm feels weak and awkward for several hours. He has abortive attacks during which the hand becomes numb and the arm jerks, without loss of consciousness. Frequently he can arrest severe attacks by placing a rubber band tightly around the left arm. For the past four years he has had a constant feeling of numbness in the left hand. He does not have headache.

Examination.—The neurologic examination was negative except that he had impaired tactile sense of the left hand and decrease of abdominal and cremasteric reflexes on the left side. The fundi were normal. There was no telangiectasis on the face or body. Examination made three days later when he was complaining of indefinite general disagreeable feelings, revealed in addition a positive left Babinski and Gordon reflex and left ankle clonus. These findings were variable. The laboratory findings were: hemoglobin, 98 per cent.; leukocytes, 7,800; red blood cells, 4,480,000; blood pressure, —110 systolic, 55 diastolic; urine, normal. The blood Wassermann reaction was negative. Spinal Fluid: pressure somewhat increased, no globulin, 4 lymphocytes, negative Wassermann reaction and colloidal gold curve.

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* Read before the Minnesota Academy of Medicine, April 14, 1921.

Diagnosis.—A diagnosis of jacksonian epilepsy due to irritation of the right motor region, cause undetermined, was made and craniotomy was performed Aug. 2, 1920, by Dr. Harry Ritchie.

Treatment and Course.—Enormously dilated blood vessels were found on the pia over the right motor region, arranged in the form of an irregular circle. These were ligated in four places and the dura was closed. During the next twenty-four hours the patient had twelve convulsions of such severity that chloroform had to be administered. The following night he had two more and was then placed on two-thirds grain of luminal three times a day. Up to the present time he has had only one unconscious seizure but at about weekly intervals he has had twitching of the left arm with blurred vision lasting a few minutes.

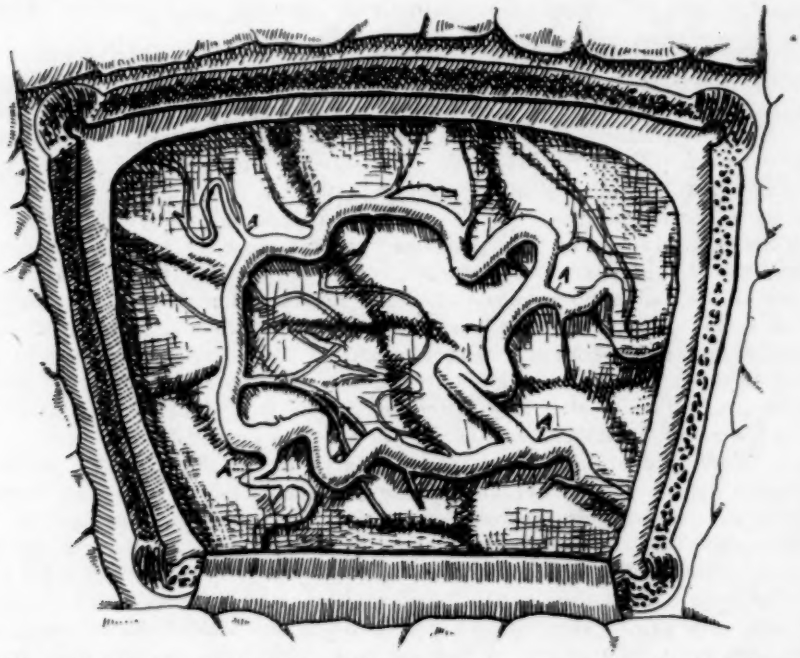

Fig. 1 (Case 1).—Jacksonian epilepsy; anterior upper margin of right ear. The vessels were ligated at *A, A, A, A;* × 1.3 +.

Comment.—Sachs reports two similar cases and believes that the clinical picture is sufficiently typical to enable one to arrive at a definite diagnosis. The outstanding symptoms are jacksonian epilepsy at long intervals in a nonsyphilitic, with unconsciousness of long duration, no evidence of intracranial pressure, slow progression of symptoms and telangiectasis on the head or face. With proper surgical interference, he makes an optimistic prognosis. Our patient conforms fairly well to this clinical picture, with one important exception—there was no evidence of superficial telangiectasis. In one of Sach's cases, the angiomatous process was found in the dura with numerous connections with the pial vessels.

CASE 2.—*History.*—Our second case was that of a farmer, 26 years old, first seen in consultation with Dr. Robert Earl, July 8, 1915. His family history was negative except that the grandfather, mother and one sister suffered from periodic headaches. His personal history was unimportant except for an occasional headache prior to the accident. In July, 1914, while running, he struck the top of his head against a protruding heating pipe. He was unconscious for thirty minutes and was sent to a hospital where he remained for two weeks, during which time he was somewhat stuporous. Recovery was complete but ten months later he developed headache in the posterior midfrontal region. This headache was progressive, more severe in the evening and so distressing that the patient threatened suicide. Within one month he developed definite mental symptoms—he thought that people were talking

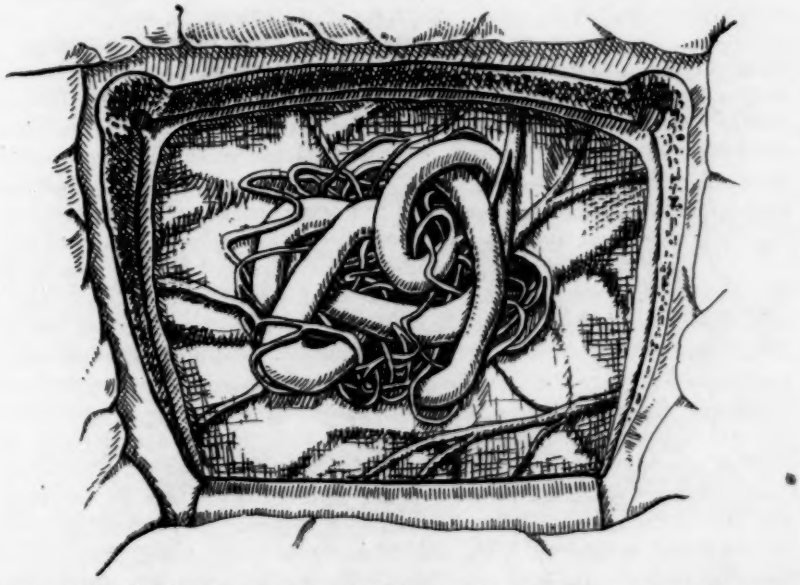

Fig. 2 (Case 2).—Multiple head injuries; inner canthus of the right eye; × 1.3 +.

about him and plotting against him and he threatened to harm them. He was also somewhat resistive and at times most unreasonable. Sleep and his general condition were satisfactory.

Examination.—Neurologic examination made one year after the accident was negative throughout except that both knee jerks were sluggish but normal on reenforcement; there was a small area of marked tenderness to pressure over the posterior portion of the right frontal region. All laboratory findings were normal. The spinal fluid was under slight pressure, the globulin test was negative, 5 lymphocytes per cubic millimeter; the Wassermann test and colloidal gold curve were negative. A roentgen-ray examination of the head was negative.

Diagnosis.—A diagnosis of beginning dementia praecox or of a cyst secondary to the trauma in the right frontal region was made.

Treatment and Course.—Because of extreme tenderness over this area, operation was advised. Dr. Robert Earl made a right frontal bone flap and found

the dura adherent to the skull. The dura appeared bluish, which was found to be due to a marked angiomatous condition on the surface of the brain cortex. This mass of blood vessels covered the entire operation field and appeared like a nest of bluish angleworms. Vessels were ligated in several places; bleeding was profuse but easily controlled. The patient made an uneventful recovery both physically and mentally.

Subsequent History.—The subsequent history is of interest. On Aug. 30, 1916, he was struck on the head by a small plank, which rendered him dizzy for a short time; after this he had occasional headaches. On June 6, 1917, he was blown up by an explosion, striking his head forcibly against a stone wall. His headaches were decidedly worse after that. On July 30, 1917, he stood for a time bareheaded in the hot sun, after which his headaches were so severe that large doses of morphin were required. Since October, 1916, spicules of bone had at times come through a discharging sinus at the site of operation. He was seen again Aug. 6, 1917. All neurologic findings were negative; the spinal fluid was normal. The old bone flap was loose at the median line and could be pressed in. On Aug. 8, 1917, Dr. Robert Earl transplanted two tibial grafts to fill the defect in the skull; the dura appeared normal except that there were extensive hemorrhages. He recovered and joined the Navy in November, 1917. In February, 1918, he was accidentally kicked on the head by another sailor and was unconscious for fifteen minutes, after which he recovered. It was found that he had a small depressed fracture over the right posterior frontal region. This depressed fracture was probably one of the tibial grafts which had become loosened by the kick. Ten days later, he developed generalized convulsions, as many as three in one week. During one of these attacks he threatened to kill the orderly with a razor. He was discharged from the Navy because of traumatic epilepsy. The skull wound discharged for about a year and then healed. During the year 1918 he had two more generalized convulsions. In July, 1919, he was again struck on the head by a small tree and was unconscious for several hours, after which he became acutely maniacal for two hours, during which time he attempted to kill one of the men with him. It required five men to put him in restraint. He recovered within an hour and remained in the hospital for two weeks when he returned to work. In July, 1920, he developed left foot drop and some frontal headaches. These continued until October when the foot drop improved, but the headache became more severe and memory was impaired. He was operated on Jan. 27, 1921, by Dr. J. A. Caldwell, United States Public Health Surgeon, at Cincinnati. The patient stated that the doctor had found depressed bone, removed it and filled the gap with a piece of rib. He has been perfectly well since that time, except for slight impairment of memory and the scars on his head. The neurologic examination on March 12, 1921, was negative except that the knee jerks were still sluggish, especially the left one.

LITERATURE ON THE SUBJECT

A true psychosis is not infrequent with cerebral lesions, especially when they involve the frontal region. Any form of mental picture may develop secondary to trauma. Under the head of post-traumatic constitution, Adolph Meyer [1] mentions paranoid states somewhat similar

1. Meyer, Adolph: Am. J. Insan., January, 1904.

to the psychosis presented by our patient. The tendency to homicidal impulses in this case, on three separate occasions, secondary to trauma, is of rather infrequent occurrence.

Both Sachs [2] and Spiller,[3] in their articles on cerebral telangiectasis, give a fairly complete review of the literature. In the majority of instances, patients presented evidences of telangiectasis on the face, head or somewhere on the body. In the discussion of Sachs' paper cases were reported by Starr, Angell, Gordon and Hunt.[4] Steiner [5] reported the cases of three families with hereditary hemorrhagic telangiectasis and reviewed twenty-five cases from the literature. The outstanding features were telangiectasis and repeated epistaxis. In none of his cases, however, was any mention made of cerebral involvement. Our two patients had no superficial telangiectasis themselves and knew of none in relatives.

Cerebral angiomas have been classed as congenital tumors, new growths or secondary to trauma. When associated with telangiectasis elsewhere, the congenital factor seems an important one. In reviewing the cases in the literature, trauma is of frequent occurrence. According to Sachs, in certain cases it seems highly probable that a trauma has either irritated a preexisting growth making it develop rapidly, or has actually been the cause of the new growth. Local inflammatory reaction, such as adhesions, connective tissue overgrowth or roughened inner layer of the dura over the dilated vessels, are further evidences of this. The history in my second case and the operative findings lead one to believe that trauma probably was an etiologic factor.

Surgery is the only treatment for these patients. Ligation of the dilated blood vessels has given surprising results in some instances. Sachs suggests that the blood vessels be ligated in such a way that no thrombosis forms, but that they collapse after ligation, thus preventing irritation of the cortex. Spiller calls attention to the danger of extensive ligation and reports two cases. In one case hemorrhage occurred into the brain and in the other hemiplegia resulted.

In our first case, the four main trunks were ligated without any serious after-effects except the series of convulsions within twelve hours after operation, in which the patient almost collapsed.[6]

435 Hamm Building.

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2. Sachs, E.: Am. J. Med. Sc., October, 1915.
3. Spiller, W. G.: Congenital Tumor of the Brain (Telangiectasis) and Associated Cerebral Movements, Arch. Neurol. & Psychiat. **2**:50 (July) 1919.
4. Nerv. & Ment. Dis., 1915, p. 634.
5. Steiner, W. R.: Telangiectasia, Arch. Int. Med. **19**:194 (Feb.) 1917.
6. In addition to the references given, the following may be of interest:
   Cassiver, H.: Neurol. Centralbl., 1902, p. 32; 1910, p. 456.
   Deetz, E.: Virchows Arch. f. path. Anat. **168**:341.
   Kalischer, S.: Berl. klin. Wchnschr., 1897, p. 1059.

THE CONTRAST BETWEEN THE BRAIN LESIONS PRODUCED BY LEAD AND OTHER INORGANIC POISONS AND THOSE CAUSED BY EPIDEMIC ENCEPHALITIS*

G. B. HASSIN, M.D.

Associate Professor of Neurology, University of Illinois College of Medicine; Histologist, Illinois State Psychopathic Institute; Attending Neurologist, Cook County Hospital

CHICAGO

Brain lesions caused by inorganic poisons, such as lead, are collectively designated encephalitis. The latter term is also used for cerebral lesions caused by infection, such as general paralysis, trypanosomiasis, epidemic encephalitis, Heine-Medin's disease and the so-called enzootic encephalomyelitis of horses (Borna disease [1]).

Best studied are the cases belonging to the infectious group chiefly represented by paralytic dementia and epidemic encephalitis. These two morbid entities exhibit such typical interstitial and parenchymatous changes that they deserve to be dignified by the term encephalitis, that is to say, inflammation of the brain tissues. Being essentially alike in their histologic changes, they differ principally in the localization of the lesions which are mainly cortical in general paralysis and mesencephalic in epidemic encephalitis.

The nonsuppurative cerebral lesions caused by organic or inorganic poisons are somewhat different from the foregoing group, the difference being, in some respects, striking enough to permit a differential diagnosis under the microscope.

This contribution deals with a short review of brain changes resulting from two inorganic poisons—lead and arsenic. The changes produced by lead poisoning have been studied in three cases which were followed up clinically and confirmed by chemical postmorten examinations. The arsenical changes have been studied in photomicrographs and specimens from experimental encephalitis which Dr. A. Jacob of

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* From the pathology laboratories of Cook County Hospital, and Illinois State Psychopathic Institute.

* Presented before the first annual meeting of the Association for Research in Nervous and Mental Diseases, New York, Dec. 28-29, 1920.

1. Joest, E.: Untersuchungen über die pathologische Histologie, Pathogenese und post mortale Diagnose der seuchenhaften Gehirn-Rückenmarksentzündung (Bornaschen Krankheit) des Pferdes, Deutsch. Ztschr. f. Nervenheilk. **42**: 293, 1911; **44**:206, 1912.

Hamburg produced in monkeys by intraspinal arsphenamin injections. He kindly placed at my disposal some of the stained sections and a great number of his photomicrographs.

Lead poisoning is usually a chronic or subacute disease process, but it seems desirable to contrast the pathologic changes occurring in epidemic encephalitis with those observed in *acute* lead poisoning. Of the three cases studied two were examples of plumbism in painters, of rather prolonged duration (about ten months), while the third was an acute lead poisoning which, because of unusual clinical phenomena, will be recorded briefly.

Fig. 1.—Cortex, motor area (lead encephalitis), and pia-arachnoid which is thickened and infiltrated. Some of the infiltrating cells are reproduced in Figure 6. Toluidin blue; × 120.

CLINICAL HISTORY OF AN ACUTE CASE

History.—March 29, 1920, a German housewife, 38 years old, mother of five healthy children, was admitted, in a delirious condition, to the neurologic service of Cook County Hospital. It was learned from her husband that she had always been well, had had no miscarriages, had never worked in a factory and presumably had nothing to do with lead. About five weeks before admission she was taken sick with pain in the chest and a cough. A physician prescribed medicine which she expelled by vomiting. She vomited after each

meal for several days. The vomitus consisted of the food taken and did not contain blood or coffee ground masses. She was in bed for three days. Ten days prior to her entrance to the hospital she was seized with severe abdominal pain, vomiting, excessive thirst, headache and backache. For several days she complained of seeing double, and on the day before admission she became delirious.

Examination.—When admitted to the hospital the patient was disoriented and talked gibberish with but an occasional understandable phrase. The speech was tremulous, the skin pale, lemon-yellow in color. There was marked pyorrhea, and a characteristic blue line was seen along the edges of the gums.

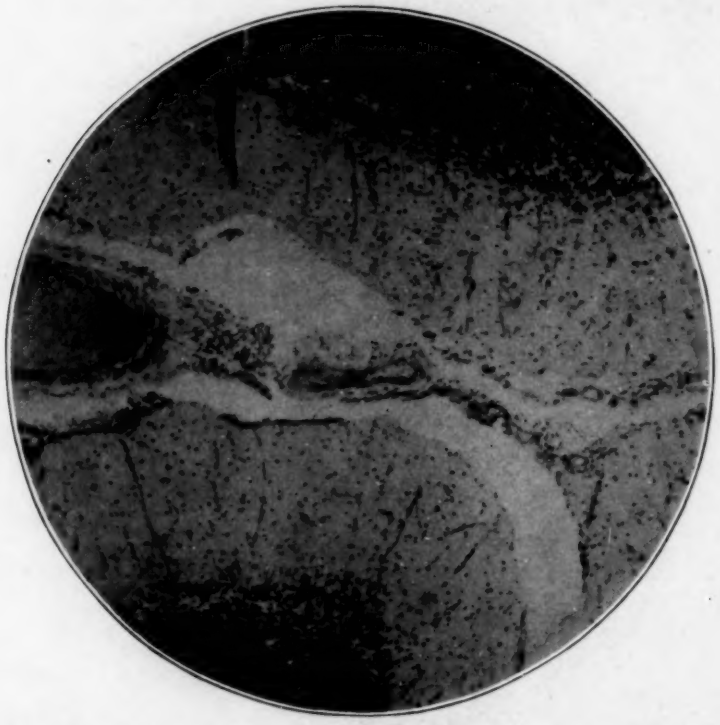

Fig. 2.—Cerebellum and its pial prolongations (lead encephalitis). The molecular layer of the cerebellum shows an abundance of tender, newly formed vessels and numerous glia cells described in the text; the pia is infiltrated with hematogenous elements shown (partly) on photomicrograph 7. Toluidin blue; × 65.

The tongue was heavily coated and the lips were covered with sordes. Nothing abnormal was discovered in the heart, lungs or abdominal organs. Paralyses, atrophies and trophic skin lesions were absent.

The pupils were equal, irregular and reacted sluggishly to light; there was an occasional nystagmus and a tremor around the mouth during talking. Otherwise the cranial nerves appeared normal. The tendon reflexes were normal; the abdominal reflex was absent; and the plantar reflexes were normal without Babinski or Oppenheim phenomena.

The spinal fluid was under low pressure, with 112 lymphocytes per cubic millimeter. The Lange and Wassermann tests were negative. The blood gave a negative Wassermann reaction and counts revealed 2,340,000 red cells and 17,900 white cells per cubic millimeter: Micro-, macro-poikilocytes and normoblasts were present but no basophilic red cells.

Differential count revealed: small mononuclears 4 per cent., large mononuclears 15 per cent., and polymorphonuclears 70 per cent. The urine was of dark amber color and contained traces of albumin with many granular casts and epithelial cells. The temperature, normal at the time of admission, was

Fig. 3.—Cerebellar pia (lead encephalitis), thickened and hyperplastic with an abundance of vessels and cellular elements. Perivascular infiltrations are lacking. Combined Bielschowsky-Alzheimer-Mann stain; × 150. Compare with Figure 16.

during the patient's four days' stay in the hospital 103 and on the day of her death 104 F.

The main macroscopic findings at necropsy, as recorded by the pathologist of the Cook County Hospital, were: marked so-called lead line of the gums (lead poisoning), marked fatty changes in the liver, icterus, acute generalized emaciation, edema of the leptomeninges, acute simple perityphlitis, hyperemia of the bowels, hyperemia of the gastric mucosa and slight edema of the lungs.

Diagnosis.—The diagnosis of lead poisoning was made during life and was based on the presence of the characteristic lead line on the gums with severe

abdominal pain, vomiting and anemia. No history of exposure to lead was obtained at first, but later it was learned that "she drank some lead (by mistake?)."

Comment. — Certain features in the clinical history are unusual in lead poisoning, namely, fever, diplopia and sluggish pupils. These symptoms might raise the suspicion of a delirious type of epidemic encephalitis. The fact of lead poisoning, however, appeared to be fully substantiated and the histologic findings, to be described, are identical with those which have been described by others both for experimental and accidental plumbism. They are in no

Fig. 4.—Optic chiasm (lead encephalitis). The parenchyma is infiltrated with connective tissue and vessels. Alzheimer-Mann stain; × 60.

way different from those made in the other two cases. The only reason for describing in detail this case in which there might be raised the question of diagnosis is that the poisoning was acute and hence more exactly comparable with epidemic encephalitis. As will be seen from the description which follows, the microscopic changes are entirely different from those of the latter disease. The facts thus afford a striking example of the possibility of differentiating between the two forms of encephalitis by histopathologic studies.

HISTOLOGIC STUDY

The changes described herewith and the illustrations are taken from Case 3 but are identical with those from the two more chronic cases. They correspond

also with the changes described as lead encephalitis by Mott[2] and Bonfiglio.[3]

1. *Pia-Arachnoid and Subarachnoid Space.*—The pia-arachnoid (Figs. 1, 2, 3 and 4) was thickened and hyperplastic. There were numerous blood vessels and the distended meshes contained many scattered cellular elements, most of them ill defined in character. Present all over the cortex, the pia-arachnoid changes were especially marked on the base of the brain and in the region of the cerebellum and optic chiasm. As seen in Figure 4, the parenchyma of the latter was invaded by large strands of hyperplastic connective tissue, derived from the pia, which consisted mainly of thickened and obliterated vessels. The

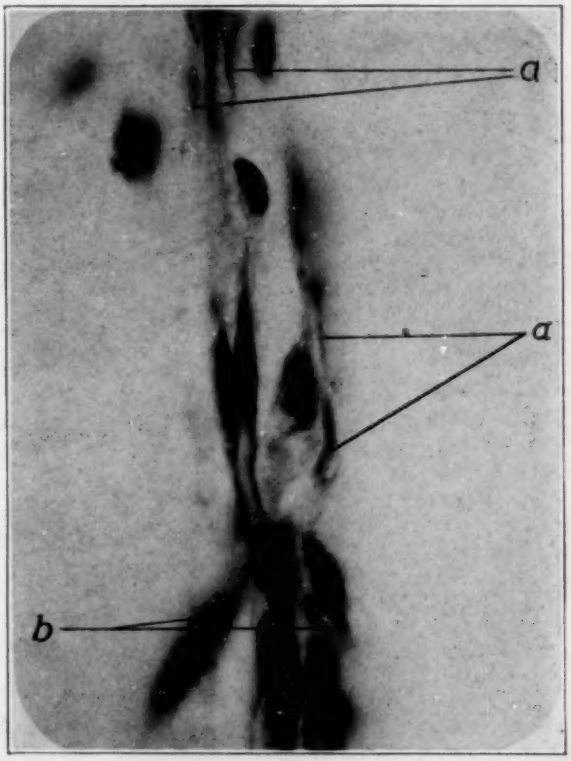

Fig. 5.—Cerebellar pia (lead encephalitis). Proliferated capillaries. Hypertrophy of the endothelial cells, their elongation, the buddings (B) are very marked. A A A, adventitial cells; the rest are endothelial cells. The adventitial spaces, especially to the right, between the adventitial cells (A) and the large endothelial cells are quite marked. Toluidin blue; ×1200.

latter were also quite abundant in the cortical pia and its intracerebral and intracerebellar prolongations (Fig. 3). These revealed a wealth of newly

2. Mott, F. W.: Examination of the Nervous System in a Case of Chronic Lead Encephalitis, Arch. Neurol. & Psychiat., London **4**:117, 1909.

3. Bonfiglio, F.: Circa la alterazioni della cortecia cerebrale consequenti ad intossicazione sperimentale da carbonato di piombo (Encephalite productiva), Nissl-Alzheimer's Arb. **3**:359, 1909.

formed blood vessels and capillaries (Fig. 5) surrounded by proliferated fibroblasts which were frequently packed with greenish or dark granules.

The endothelial cells of the capillaries (Fig. 5) were well developed, hypertrophied, brightly stained and rich in chromatin. Frequently they showed signs of proliferation and budding. There was no perivascular infiltration though there were sometimes numerous cellular elements of various types scattered among the vessels. Many of these cells (Fig. 6) were dark, round and deeply stained and some of them on closer examination showed numerous minute granules and a pale rim of cytoplasm. Some cells were rather broken up and appeared ragged, either freely scattered in the pial meshes or gathered near

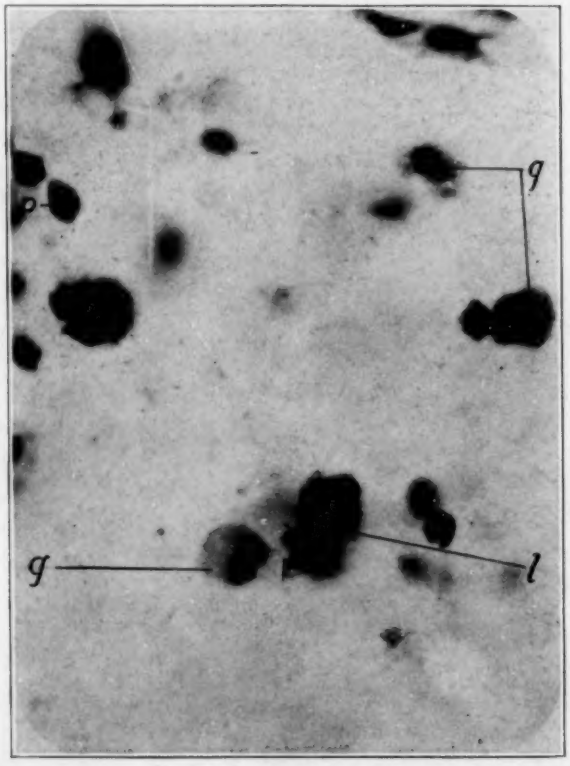

Fig. 6.—Some of the cells of the cortical subarachnoid space (lead encephalitis). The majority of these are modified lymphocytes. The large reticulated protoplasmic bodies are probably gitter cells; some are degenerated plasma cells, polyblasts and clusters of lymphocytes. P, plasma cells; G G, gitter cells; L, cluster of lymphocytes. Toluidin blue; × 1200.

the blood vessels in the form of clusters. These were evidently modified lymphocytes or plasma cells. In some places they possessed a large amount of homogeneous, metachromatically stained cytoplasm. This, in some instances, appeared granular or reticular, surrounding a pale, oval, eccentric nucleus and the cells thus very much resembled the so-called "gitter" cells. The former type, with homogeneous cytoplasm, may be classified as polyblasts, or large mononuclear lymphocytes. In addition to these three types, lymphocytes, poly-

blasts and gitter cells, there were a great many fibroblasts, as well as large, oval, pale bodies (Fig. 7, A) containing a few chromatin granules, located singly or in clusters. These were most probably the so-called cover arachnoid cells ("Belegzellen") usually described as mesothelial cells (Weed, Essick). The masses of cellular elements, the abundance of vessels and the distended pial meshes caused the meninges to appear greatly thickened (Fig. 3). On the base, especially around the cerebellum, the foregoing elements were very numerous, sometimes forming dense foci (Fig. 7) consisting of well preserved lymphocytes, plasma cells and many other less typical cell bodies. Such dense foci, however, were present only in the acute case and were much less in evidence in the other two. The foregoing cells did not invade the parenchyma of the cerebellum, as in epidemic encephalitis, but were strictly confined to the pia, its meshes, and the neighborhood of vessels. In the region of the optic chiasm the cellular infiltration was either entirely lacking or comparatively

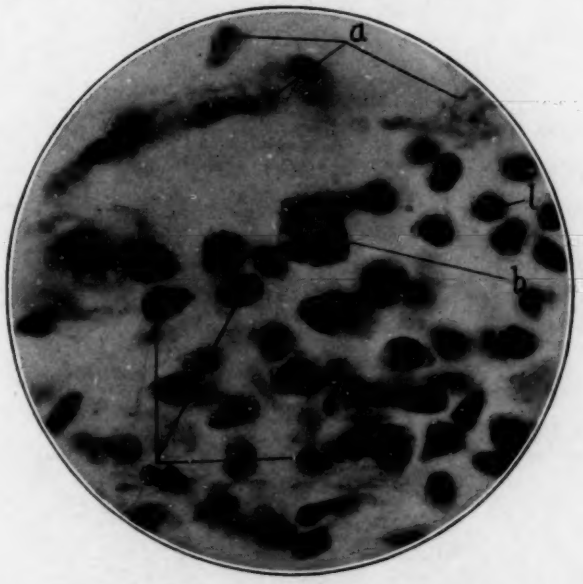

Fig. 7.—Focus of infiltration of the cerebellar subarachnoid space (lead encephalitis). A, mesothelial cells of the arachnoid, hypertrophied; B, large plasma cell; C, smaller plasma cells; L, lymphocytes. Other cells are less well defined. Toluidin blue; × 1200.

mild, being replaced, as noted, by an abundance of connective tissue and vessels which in some places completely obliterated the invaded parenchyma.

In the spinal cord (only the upper cervical portion was available) the subarachnoid space and the pia showed the presence of pigment, lymphocytes and other elements, mostly ill defined.

Summary of Pia-Arachnoid Changes: There were progressive or proliferative changes, especially pronounced on the base of the brain, around the cerebellum and in the optic chiasm. Aside from the marked proliferative or hyperplastic phenomena, there were also present in some areas, especially in the acute case, pure inflammatory changes in the form of infiltration with lymphocytes and plasma cells.

2. *Parenchymatous Changes.*—The various regions of the cerebral cortex, the basal ganglions, midbrain and cerebellum have been studied with toluidin blue, thionin, Herxheimer's scarlet red and other methods.

The architecture of the cortical layers in the various regions was well preserved and easily recognized. The subpial zone (stratum zonale) of the cerebral cortex and the molecular layer of the cerebellum (Figs. 1 and 2) were covered with abundant glia cells well supplied with cytoplasm and thin, pale, ramifying processes. Their nuclei were quite large and rich in chromatin, while the ramifying processes could in some instances be followed for a long distance, sometimes even reaching the walls of the capillaries. In the cerebral white substance (Figs. 8 and 9) the glia cells were mostly represented by nuclei, often gathered in rows along the smaller vessels and capillaries. They

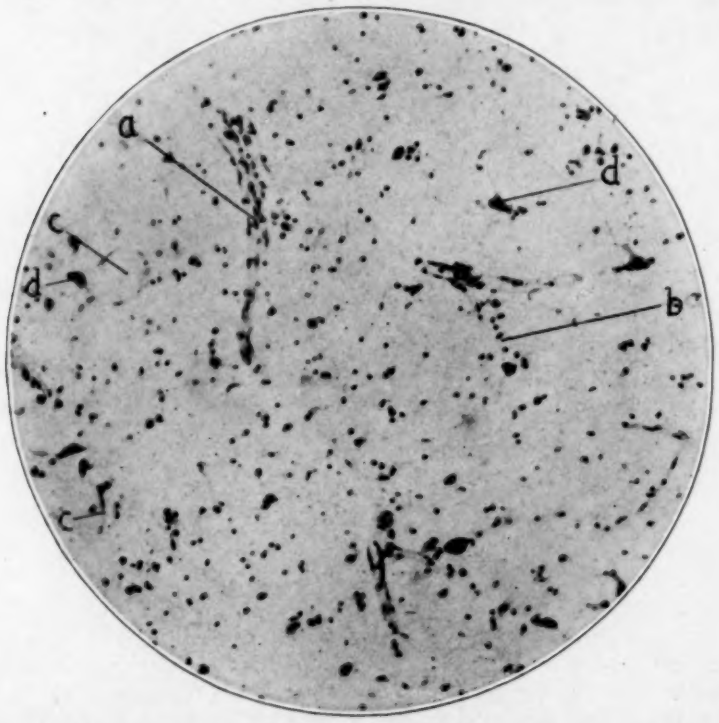

Fig. 8.—Midbrain (region of sylvian aqueduct) fom a case of lead encephalitis. Three prominent capillaries. At A, the upper portion shows two parallel capillaries; at B, a row of glia cells is well marked. The endothelial cells, even in the smallest capillary C, are prominent. D, ganglion cells. The numerous black dots are glia cells in which with the help of a hand lens protoplasm can be seen. Toluidin blue stain; × 130. Compare Figure 14.

were always situated outside the adventitial space and frequently formed clusters. More complicated gliogenous formations, such as myeloblasts, myelophages, and the various types of gitter cells could not be found. Stained with scarlet red, the glia cells sometimes showed fat in the form of minute globules (Fig. 10). In the white cerebral substance the glia cells were numerous, and in some places the glia tissue appeared reticulated.

The ganglion cells appeared normal, though their processes frequently were diffusely stained and tortuous and could be traced for a long distance. The cytoplasm was homogeneous, often broken up (chromatolysis), the nucleus eccentric with well preserved membrane and chromatin substance.

Some cells, especially those of the deeper layers, were densely surrounded and even invaded by glia cells (satellitosis and neuronophagia). In some areas, for example, the cerebellum, the ganglion (Purkinje) cells showed marked vacuolation (Fig. 11), while in the midbrain the ganglion cell changes were almost lacking. Stained with scarlet red, the ganglion cells uniformly and universally showed the presence of fat in the form of large globules within the cell body and its processes but the nucleus was left intact (Fig. 10)

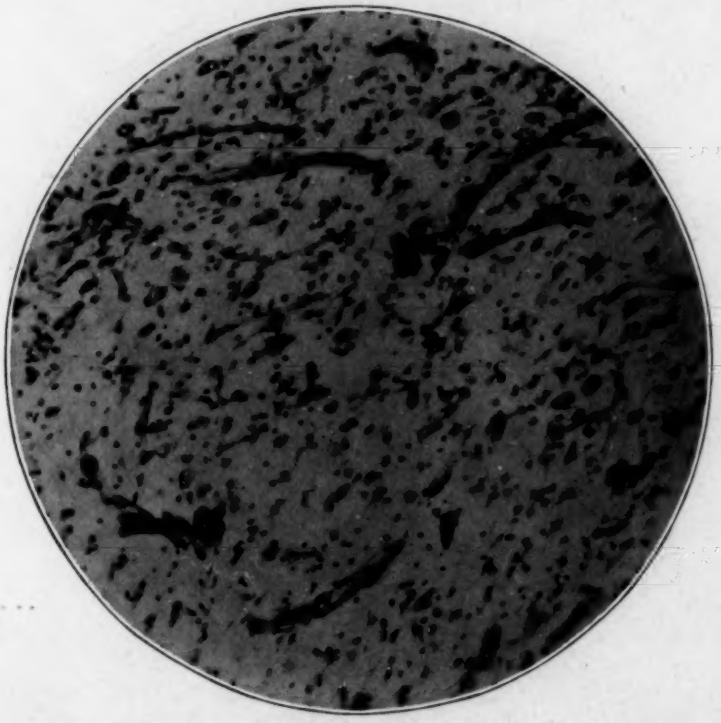

Fig. 9.—Temporal lobe (lead encephalitis). The entire field is covered with an abundance of proliferated blood vessels containing prominent endothelial and adventitial cells. The contrast with Figure 15 is marked. The abundance of glia nuclei is striking. Toluidin blue; × 130.

The nerve fibers of the brain and spinal cord (cervical region), stained with Bielschowsky or Alzheimer-Mann methods, did not appear changed; sometimes they were somewhat thickened and tortuous, especially in the subcortical area, but there were no signs of degeneration or inflammation.

Summary of Ectodermic Changes: The glia cells, like the pia-arachnoid, showed marked progressive changes; increased size of nuclei, abundance of chromatin and cytoplasmic processes and a great proliferation of glia nuclei gathered in rows or clusters. They usually contained fat and frequently

invaded the ganglion cells. Most of the latter were well preserved, some showing chromatolytic changes, vacuolation and neuronophagic phenomena.

3. *Vascular Changes.*—The cortex, subcortical white substance, large ganglions, midbrain, cerebellum, choroid plexus, meninges and spinal cord (cervical region) all showed an excessive vascularization. The smallest capillaries were much in evidence, being usually engorged with blood and exhibiting unusually well stained endothelial and adventitial cells. Some capillaries were exceedingly small and narrow and contained no blood cells, often being represented by a few proliferated endothelial or adventitial cells or by offshoots, buddings or slight protrusions of a vessel wall.

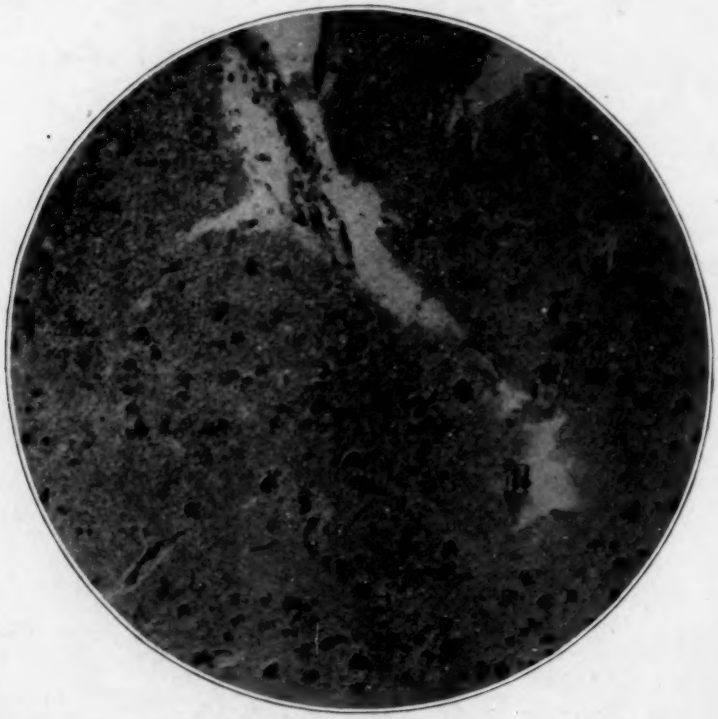

Fig. 10.—Temporal lobe. The ganglion cells, many glia cells, and especially the blood vessel (the upper part of the picture), show fat globules in the form of black spots. Herxheimer scarlet red; × 150.

The appearance of the endothelial cells, rich in chromatin, brightly stained and with cell body elongated as if thinned out, is well shown on the accompanying photomicrographs (Figs. 5, 12 and 13). The budding formations and the endothelial changes, as well as the general appearance of some newly forming capillaries, are especially well seen in Figure 5. Similar, and even more striking, new vessel formations may also be seen in abundance in the cerebral and cerebellar pial prolongations, in which they are found in every stained specimen. In fact, newly formed capillaries predominate and are much more numerous and conspicuous than fully developed preformed vessels.

In some smaller vessels and capillaries the adventitial and endothelial cells were distinctly arranged in the form of membranes separated by an adventitial space (Fig. 5) which, however, were nowhere infiltrated. The endothelial, as well as the adventitial, cells were frequently filled with greenish granules; and, as already noted, in the cerebellar pial prolongations they were densely surrounded by lymphocytes, plasma cells and other hematogenous elements.

The walls of the larger vessels were appreciably thickened, the thickening being due to proliferation of the adventitial cells and a great accumulation of fibroblasts, some of which were packed with pigment granules. The endothelial cells, as in the smaller vessels, were either increased in size with a nucleus rich in chromatin or they appeared to have proliferated and formed clusters and buddings.

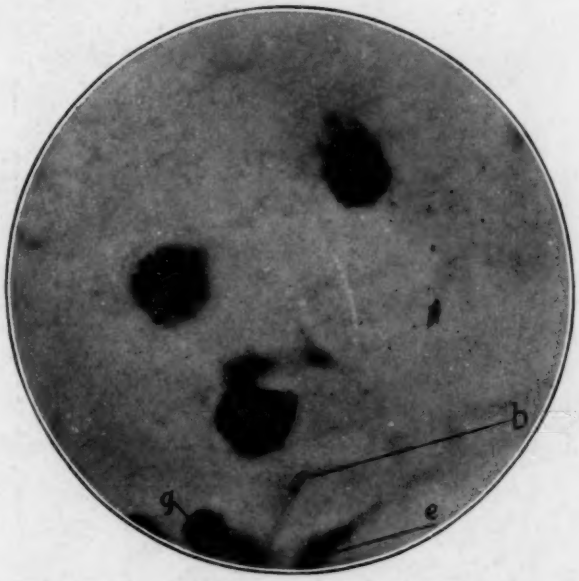

Fig. 11.—Three changed ganglion cells from the cerebellum: the nuclei are eccentric but well preserved. The cell bodies, only partly reproduced, appear pale and vacuolated. Below is a capillary with prominent endothelium (E) and budding (B); near by is a glia cell (g) well supplied with cytoplasm. Use a hand lens. Thionin; × 1200.

In the molecular layer of the cerebellum many so-called rod cells ("Stäbchenzellen") were seen. Some of them were sausage-like in shape, as if about to form a capillary, while others had visible processes emanating from a densely stained, chromatin-rich nucleus. These cells were seen only in toluidin-blue or thionin stained specimens and were absent from those stained with the Alzheimer-Mann method. In the latter they appeared as young capillaries or adventitial cells.

Like the capillaries and smaller blood vessels, the larger ones did not exhibit perivascular infiltrations. Neither was the surrounding parenchyma invaded by blood elements (lymphocytes, plasma cells), as is often the case in epidemic encephalitis and general paralysis.

Figure 8, for instance, shows the condition of the midbrain in lead encephalitis, while Figure 14 presents the corresponding area in the epidemic type. The latter is densely infiltrated, while the former exhibits only proliferating vessels.

Summary of Vascular Changes: The vessels showed marked proliferative or progressive phenomena, as evidenced by endothelial and adventitial proliferation and newly formed capillaries. Signs of perivascular infiltration, such as is seen in epidemic encephalitis, were lacking.

ARSENICAL POISONING

The findings described above for the three cases of lead poisoning are exactly similar to those found in the specimens and photographs

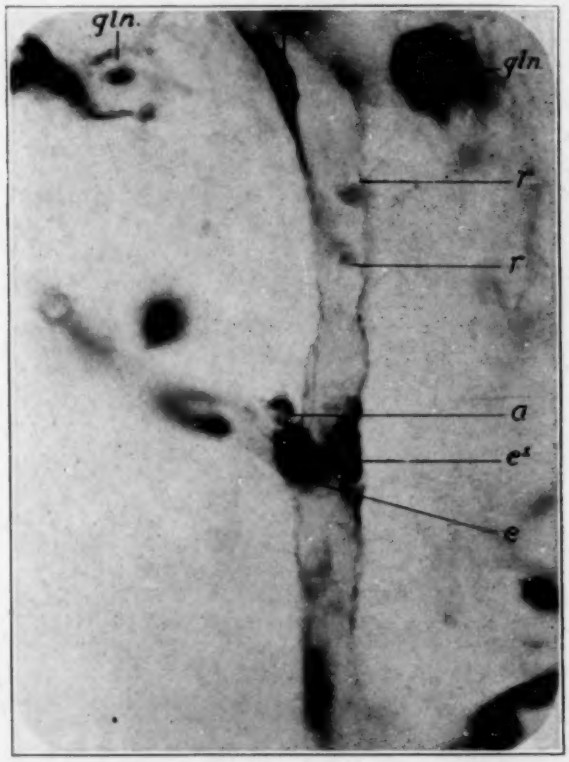

Fig. 12.—Newly formed capillaries from the cerebellar cortex E E_1E_2, endothelial cells; at E a budding formation is distinctly seen; A, adventitial cell with a cut off process (use lens); R R, red cells; Gln, ganglion cells. Toluidin blue, × 1200.

furnished to me by Jacob, following experimental arsphenamin poisoning in monkeys. These have been described by Jacob,[4] and therefore need not be further detailed here.

4. Jacob, A., Weygand, W., and Kafka, V.: Klinische und experimentelle Erfahrungen bei Salvarsaninjektionen in das Zentralnervensystem, München. med. Wchnschr. **61**:1608, 1914. Die Entzündungsfrage in Zentralnervensystem (Allgemeine Uebersicht), Jahreskurse für ärztliche Fortbildung, May, 1919.

GENERAL SUMMARY AND DISCUSSION

The changes produced in the brain tissues by inorganic poisons (lead, arsenic) are both parenchymatous and interstitial.

The former, degeneration of ganglion cells with accumulation of lipoid substances, are essentially regressive or degenerative. The interstitial changes, on the other hand, are proliferative or progressive in

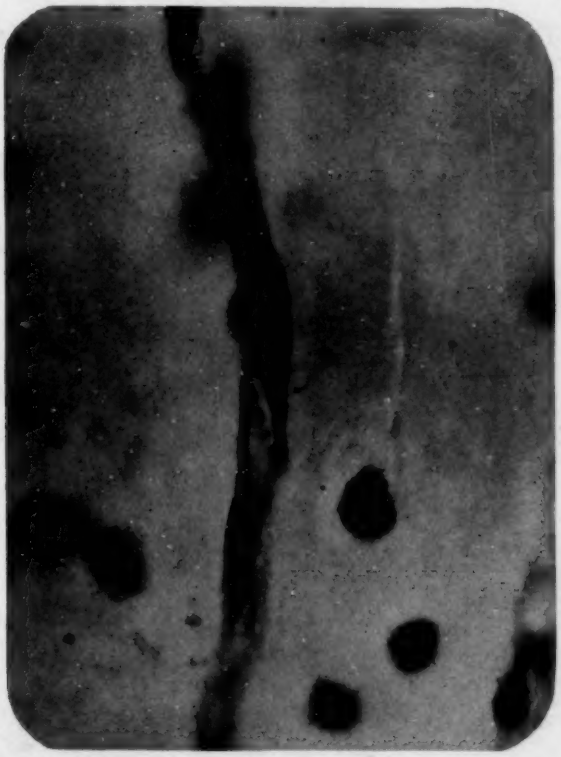

Fig. 13.—A newly formed capillary (lead encephalitis). The vessel is made up of parallel endothelial cells which contain chromatin-rich nuclei with an appreciable amount of cytoplasm at their poles. The cytoplasm is thinned out forming the capillary walls. The dark bodies outside the capillary are enlarged protoplasmic glia cells packed with chromatin. Thionin; × 1200.

character (glia cell proliferation, progressive phenomena in the endothelial and adventitial cells, new formation of capillaries).

While the parenchymatous changes are not peculiar or typical but resemble those to be found in any type of encephalitis, including that of the epidemic variety, the mesodermal changes of the pia and vessels

are characteristic and essentially different from those seen in epidemic encephalitis. In the latter, the vascular changes are distinctly infiltrative in character and are only occasionally present in the pia (Figs. 15 and 16).

In lead encephalitis the infiltrative features are either altogether absent or are insignificant; in fact, they are to be found only in acute or peracute cases like the one in the foregoing. Instead, we find marked proliferations in the vessels as well as in the pia-arachnoid. These pro-

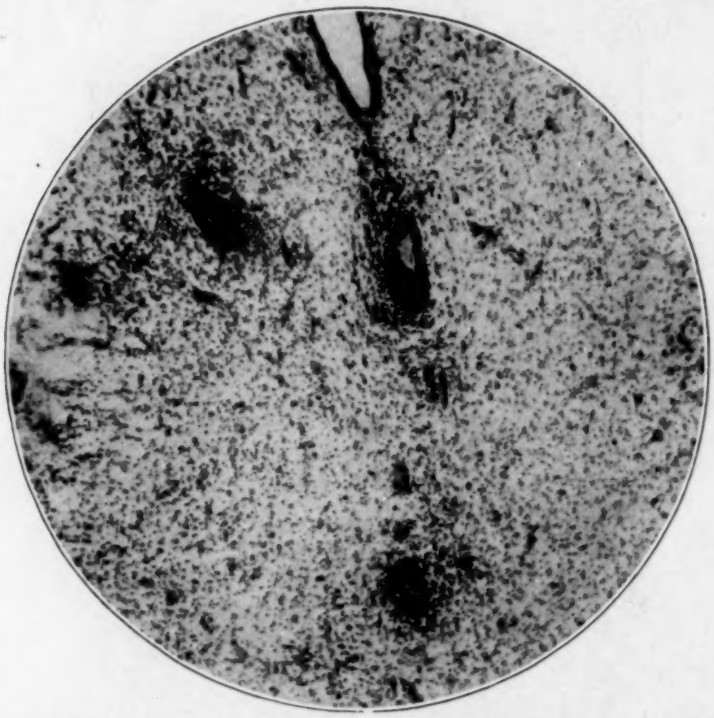

Fig. 14.—Midbrain (region of aqueduct of Sylvius) from a case of epidemic encephalitis. The vessels, as well as the parenchyma, are densely infiltrated with lymphocytes and plasma cells. Compare with Figure 8. Toluidin blue; ×65.

liferative changes are universal but they are especially marked on the base of the brain and around the cerebellum, optic chiasm and temporal lobe. They are comparatively slight in the midbrain. The lesions in epidemic encephalitis, by contrast, always involve, though not exclusively, the midbrain.

The prevalence of productive or proliferative phenomena in experimental lead encephalitis was also noted by Bonfiglio, who suggested for this type the descriptive designation "productive." In contradistinction

to this, we might label the infectious type (to which also belong numerous other forms of nonsuppurative encephalitis caused by various infections, paralytic dementia, Heine-Medin's disease, rabies, pernicious malaria, trypanosomiasis, Borna disease) "infiltrative." It thus seems permissible to claim that vascular infiltrative phenomena are indicative of an infectious type of encephalitis, while the proliferative phenomena denote a toxic condition.

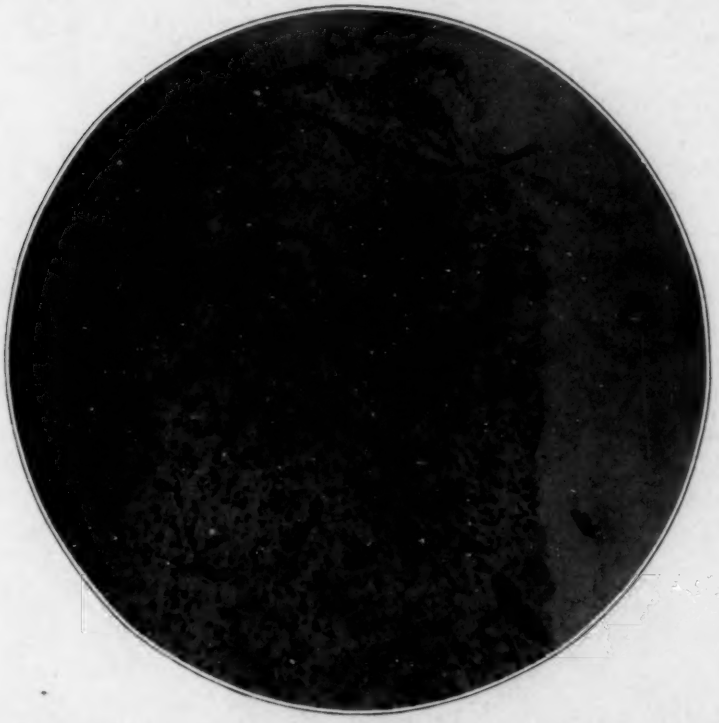

Fig. 15.—Cortex and pia from a case of epidemic encephalitis (Bassoe and Hassin). The pia-arachnoid is mildly infiltrated; the cortex shows a few infiltrated vessels, especially in the deeper layers. Compare with Figure 9. Toluidin blue; × 50.

Proliferative and infiltrative phenomena should be sought not only in the endothelial, adventitial and other vascular tissues of the brain proper, but also, and even with greater care, in the pia-arachnoid and the subarachnoid space.

In toxic encephalitis numerous, more or less ill-defined, cellular elements are mixed with mesothelial arachnoid cells, often in the form of clusters. This is not the proper place to discuss the origin and character of the various pial cellular elements, but it may be pointed out that cluster formation or proliferation of the mesothelial arachnoid

cells is decidedly pathologic, for they represent a reaction against the invasion of the subarachnoid space by some irritating foreign substance. In the case of lead encephalitis this substance is some toxic product. Essick,[5] as well as Weed,[6] distinctly showed that the mesothelial cells react against irritants, such as particulate matter, and are capable of being transformed into new cell bodies resembling macrophages and under certain conditions, old age, for instance, even tumor-like masses (Weed).

Fig. 16.—Cerebellum and pia from a case of epidemic encephalitis (Bassoe and Hassin). The pia is infiltrated, the molecular layer densely covered mostly with hematogenous elements (lymphocytes and plasma cells). Toluidin blue; × 150. Compare with Figure 3.

The condition of the subarachnoid space and its membranes may thus be considered a valuable standard for the purpose of differentiating various types of brain lesions, whether purely inflammatory, degen-

5. Essick, Charles R.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter. Contributions to Embryology, No. 42, Publication 272 of the Carnegie Institution of Washington, pp. 377-388.

6. Weed, L. H.: The Cells of the Arachnoid, Bull. Johns Hopkins Hosp. **31**:343 (Oct.) 1920.

erative or proliferative. In fact, they may show changes even when the brain tissues proper appear quite unaffected.

CONCLUSIONS

1. The nonsuppurative inflammations of the brain may be divided into two large groups. One, represented by epidemic encephalitis and paralytic dementia, is of infectious origin. Pathologically, it is characterized by excessive, widespread perivascular, and sometimes also parenchymatous, infiltrations, the pia-arachnoid changes being quite mild. This type may also be defined as an infiltrative encephalitis which thus includes all possible direct infections of the brain tissue proper.

2. The second group, mainly represented by lead encephalitis, is due to intoxication and should, therefore, be termed toxic. Pathologically, it shows a great preponderance of proliferative or (as Bonfiglio puts it) "productive" phenomena in the mesodermic tissues (blood vessels, capillaries and the pia-arachnoid). This group may be termed productive encephalitis.

3. Microscopically, it is not only possible to determine whether any given case is one of a nonsuppurative encephalitis, but also whether the encephalitis is due to an infection or to an intoxication.

4. The study of the pia-archnoid and the subarachnoid space is of paramount importance in determining whether the brain is normal or pathologic, for these structures may exhibit changes even when the brain tissues proper appear normal.

STUDIES IN ASYMPTOMATIC NEUROSYPHILIS

I. A TENTATIVE CLASSIFICATION OF EARLY ASYMPTOMATIC NEUROSYPHILIS *

ALBERT KEIDEL, M.D., AND JOSEPH EARLE MOORE, M.D.
BALTIMORE

Although we are as yet ignorant of the exact route followed by the *Spirochaeta pallida* in its invasion of the central nervous system, we know definitely that in many cases this invasion occurs with the generalization of the infection preceding secondary manifestations. In other words, the onset of neurosyphilis coincides in most instances with the onset of syphilis. Apparently the only exceptions are those instances in which central nervous system invasion takes place during subsequent periods of generalization of the infection, such as are believed to precede recurrent outbreaks of a secondary type; or by direct extension from an intimately associated focus of syphilitic bone or vascular disease. We may conclude that in untreated or inadequately treated cases more than one invasion of the central nervous system may occur. Ample proof of the early invasion may be found in studies of the cerebrospinal fluid in primary and early secondary syphilis.[1] In early untreated syphilis, from 30 to 50 per cent. of the fluids show more or less abnormalities, although most workers have found that only from 1 to 4 per cent. of such fluids have a positive Wassermann reaction. By means of routine lumbar puncture, therefore, it is possible in primary and early secondary syphilis to differentiate four groups of cases: one in which the fluid is normal; a second with minimal fluid abnormalities, such as slight increase in the globulin content and cell count; a third in which minimal abnormalites are associated with a positive Wassermann reaction when large amounts of fluid are used, and with a syphilitic zone gold curve or mastic "3"

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* From the Syphilis Department of the Medical Clinic, Johns Hopkins Hospital.

* Read at the Annual Meeting of the Medical and Chirurgical Faculty of Maryland, April 28, 1921.

1. Fordyce, J. A.: The Diagnostic and Prognostic Significance of Spinal Fluid Findings in Syphilis, Med. Rec. **91**:927 (June 2) 1917. Ellis, A. W. M., and Swift, H. F.: J. Exper. Med. **18**:162, 1913. Wile, U. J., and Stokes, J. H.: Involvement of the Nervous System During the Primary Stage of Syphilis, J. A. M. A. **64**:979 (March 20) 1915. McIver, Joseph: The Spinal Fluid in Primary and Secondary Syphilis, ibid. **73**:1765 (Dec. 6) 1919. Wile, U. J., and Hasley, C. K.: Involvement of Nervous System During the Primary Stage of Syphilis, ibid. **76**:8 (Jan. 1) 1921.

reaction; and a fourth group with maximal positive findings in all tests. In the first group fall the cases which presumably escape central nervous system invasion. It must be emphasized, however, that an unknown though small percentage of these may show gross abnormalities on subsequent investigation, particularly if treatment is omitted. This point is emphasized by the investigation of Steiner,[2] who with Mulzer showed that with three of twenty spinal fluids from patients with early syphilis positive inoculations in animals were obtained, although these three fluids were otherwise entirely normal. In the second group, an undetermined number are probably normal, the globulin and cell increase being irritation phenomena, comparable to the slight transient albuminuria and the appearance of a few casts so frequently observed in the urine of patients with secondary syphilis, without demonstrable kidney damage. This group, nevertheless, must be considered to contain a high percentage of cases with recent neurologic invasion, in which tissue reactions are still in abeyance or only slightly developed. The third group contains principally cases of early meningovascular neurosyphilis with possibly a small proportion of cases with incipient parenchymatous or central involvement. All cases falling in the fourth group, with all tests positive, are obviously neurosyphilitic.

DIAGNOSIS AND TREATMENT OF NEUROSYPHILIS

Although the diagnosis of early asymptomatic neurosyphilis depends on information derived from spinal fluid studies only, it is interesting to note in these patients, as reported by one of us,[3] the prevalence of persistently positive blood Wassermann reactions in treated cases and the incidence of minor pupillary abnormalities—headache, lassitude, rheumatic pains and nervousness. Of a series of 642 routine spinal fluid examinations, 173 fluids were from patients presenting these manifestations (Table 1), not of themselves diagnostic of neurosyphilis, with an abnormality incidence of 28.3 per cent., while in the remaining 469 only 7.3 per cent. presented abnormalities. In this series treatment had been instituted before the lumbar puncture was done, and, as will be shown, had a modifying influence on the results.

The factor of a persistently positive blood Wassermann reaction as an indication of asymptomatic neurosyphilis may be shown by a regrouping of the results obtained in primary and secondary syphilis (Table 2). In twenty-two cases in which the blood Wassermann reaction was still positive after six doses of arsphenamin, six or 27.2

2. Steiner: Impfexperimente mit Spinalflüssigkeit von syphilitikern, Neurologische Centralbl. **33**:132, 1914.

3. Moore, J. E.: The Cerebrospinal Fluid in Treated Syphilis, J. A. M. A. **76**:769 (March 19) 1921.

per cent. of the spinal fluids were abnormal. When the blood Wassermann reaction had become negative by the end of the first course of treatment, only 15.3 per cent. of the fluids were abnormal. If, after two or more courses of treatment the Wassermann reaction was still persistently positive, 38.4 per cent. of the fluids were positive; if negative, only 7.8 per cent. showed abnormal fluids. This shows, furthermore, that the prompt disappearance of a positive blood Wassermann reaction is not a guarantee against the existence of asymptomatic neurosyphilis.

Treatment as a preliminary to the study of spinal fluids in early syphilis was adopted by us a number of years ago, principally because we felt that more information could thus be gained. If the early minor abnormalities in the fluid were an expression of the general infection

TABLE 1.—SPINAL FLUID ABNORMALITIES IN PRIMARY AND SECONDARY SYPHILIS AS COMPARED WITH BLOOD WASSERMANN REACTION AND TREATMENT

Blood Wassermann Reaction	Cerebrospinal Fluids			
	Total Number of Fluids Examined	Positive	Negative	Percentage Abnormal
After one course of treatment:				
Positive	22	6	16	27.2
Negative	39	6	33	15.3
Total	61	12	49	19.6
After two or more courses of treatment:				
Positive	13	5	8	38.4
Negative	102	8	94	7.8
Total	115	13	102	11.3

as an irritation phenomenon, a small amount of treatment would cause them to disappear as the general infection diminished. Under these conditions remaining abnormalities would have greater significance as indicating actual spirochetal invasion of the nervous system. That in some instances the invasion might be obscured by this amount of treatment was no objection, because, if it could be so readily obliterated, long continued treatment, our routine plan, would fully safeguard the patient. The routine treatment would not be altered because of spinal fluid abnormalities, unless their persistence indicated some alteration or addition to the therapy.

We do not advocate long delay in making the puncture nor do we believe that a single puncture should be depended on to rule out asymptomatic neurosyphilis, although negative findings in early punctures minimize the possibilities if followed by adequate treatment. In more than 100 patients in whom an early negative spinal fluid examination

was obtained, reexamination of the fluid has yielded positive results in only two. In both of these an explanation for the late invasion of the nervous system was apparent. In one it probably occurred during a fresh generalization of the disease; in the second, by direct extension from syphilitic involvement of the inner cranial table. A recent communication from one of us [4] considers this point in detail.

From the foregoing it will be appreciated that the treatment of early asymptomatic neurosyphilis is so intimately dependent on the interpretation of cerebrospinal fluid findings that the two cannot be considered separately. The choice of therapeutic measures cannot be compared in importance with early recognition. We believe, however, that adequate therapy has, at this period of the invasion, a most beneficial influence on what may follow. The incidence of neurosyphilis during the period of general invasion is the strongest argument in

TABLE 2.—Spinal Fluid Abnormalities in Secondary Syphilis as Influenced by Treatment

Amount of Treatment	Cerebrospinal Fluids			
	Total Number of Fluids Examined	Positive	Negative	Percentage Abnormal
None or very little.....	19	5	14	26.3
Six doses of arsphenamin ± mercury.....	42	9	33	21.4
Twelve doses of arsphenamin + mercury.....	37	7	30	18.9
Eighteen doses of arsphenamin + mercury..	22	2	20	9.0
Twenty-four or more doses of arsphenamin + mercury.....	31	1	30	3.2

favor of the adequate treatment of all syphilis. In addition to the data already available in the literature, a series of cases studied by us from this point of view tends to confirm this opinion.

The influence of treatment in 151 cases of primary and secondary syphilis as indicated by changes in the cerebrospinal fluid was studied (Table 2). Nineteen patients had received little or no treatment and five, or 26.3 per cent., presented fluid abnormalities; of forty-two cases following six doses of arsphenamin with or without a course of mercury nine or 21.4 per cent. presented abnormalities in the fluid; of thirty-seven patients receiving twelve doses of arsphenamin with courses of mercury, fluid abnormalities were present in seven or 18.9 per cent.; 9 per cent. of patients in twenty-two cases following eighteen doses of arsphenamin with courses of mercury had abnormal fluids; and in thirty-one patients there were only 3.2 per cent. abnormal fluids

4. Moore, J. E.: The Genesis of Neurosyphilis, Arch. Dermat. & Syph., to be published.

after twenty-four or more doses of arsphenamin with courses of mercury. In this series, then, effective therapy reduced the prevalence of asymptomatic neurosyphilis from 26.3 to 3.2 per cent. If a comparison is made between the first three groups and the last two, it will be seen that after twelve or less doses of arsphenamin and courses of mercury, the spinal fluid showed abnormalities in 21.4 per cent. while after eighteen or more doses of arsphenamin plus mercury the percentage dropped to 5.7.

CLASSIFICATION OF EARLY ASYMPTOMATIC SYPHILIS

Further investigations are under way but we feel that a preliminary observation on the classification of early asymptomatic neurosyphilis based on the type of spinal fluid abnormalities and the corresponding results of treatment is permissible. Our four groups may be further described as follows:

Group 1.—Normal fluids. Neurosyphilis is not definitely ruled out, but we have no means of predicting which cases will later show abnormalities, except by animal inoculation experiments. The great majority of patients in this group certainly remain free from late clinical or serologic evidence of neurosyphilis.

Group 2.—Neurologic damage minimal or questionable. The spinal fluid shows pleocytosis and increased globulin content, but negative Wassermann and colloidal gold and mastic tests. These findings may be in some cases the expression of meningeal irritation only, without definite tissue invasion. Patients showing this type of spinal fluid uniformly do well on routine treatment without the addition of intraspinal therapy. The routine may be that for patients without spinal fluid changes.

Group 3.—Tissue invasion moderate. The usual early complaint is headache. The spinal fluid shows cells from 10 to 100, usually less than 50; globulin + or ++; the Wassermann reaction is negative with small quantities of fluid, and either positive or negative with larger amounts; colloidal gold curve syphilitic or meningitic zone; mastic curve to 3, or, in some instances, paretic. In general this type of patient does well, both clinically and serologically, on routine treatment without intraspinal therapy. However, we believe that to obtain the best results the dosage of arsphenamin and the number of doses to a course should be increased over the routine used for patients without neurologic invasion, that the interval between doses should be decreased, and that the total amount of arsphenamin administered should be relatively greater and the total amount of mercury relatively less than in uncomplicated cases. In only one instance have we found it necessary to resort to intraspinal therapy in order to accomplish a

serologic cure. It is probable that spinal fluid changes of this type represent future meningovascular cerebrospinal syphilis, though a minority of the patients may ultimately develop parenchymatous neurosyphilis.

Group 4.—Tissue invasion definite. Complaint may be absent, or may be that of nervousness, lassitude or neuralgic pains. If careful sensory examination is omitted, neurologic abnormalities are not detected. The spinal fluid shows from 10 to 100 cells, usually more than 50; the globulin is greater in content than in the preceding two groups, ranging from + + + to + + + +; the Wassermann reaction is positive with 0.2 c.c. or less, and the colloidal gold and mastic curves are paretic. The majority of patients in this group are not serologically cured by routine treatment, regardless of alterations in the individual dose or the total amount of arsphenamin, or the interval between doses. An occasional patient may be serologically improved, but the improvement is difficult or impossible to maintain. If, after six months' treatment, no change in the intensity of the spinal fluid findings is manifest, intraspinal treatment is an indispensable adjunct to routine treatment. Even when this plan is adopted, improvement, which may be looked for in a large percentage of the cases, is slow and treatment prolonged. In all probability, this group represents future cases of parenchymatous neurosyphilis—paresis and tabes. Indeed, we have observed the development of paresis in two patients with such spinal fluid findings discovered early in the course of syphilis.

315 Professional Building.

A COMPARATIVE STUDY OF THE SUGAR CONTENT OF THE SPINAL FLUID IN DISEASES OF THE NERVOUS SYSTEM*

LEWIS D. STEVENSON, M.D.
Instructor in Neuropathology, Washington University Medical School
ST. LOUIS

This work was originally begun in an attempt to show that the sugar content of the spinal fluid is increased in lethargic encephalitis and that this finding is an important aid in the differential diagnosis of this disease, especially in differentiating it from tuberculous meningitis in children.

Foster,[1] of Boston, has recently published a paper in which he gives a series of estimations by Folin's method. His average result is 76 mg. of sugar in 100 c.c. of spinal fluid in epidemic encephalitis. In a paper read by the author before the March, 1921, meeting of the St. Louis Neurological Society, a series of seven results was quoted from the service of Dr. Foster Kennedy of Bellevue Hospital, New York, the average being 79.3 mg. of sugar per 100 c.c. of fluid in cases of lethargic encephalitis. These estimations were all made by Folin's method, and I am indebted to Dr. Kennedy for permission to make use of these figures.

The table gives a series of sugar determinations in various neurologic conditions, from the service of Dr. Sidney I. Schwab of Barnes Hospital, St. Louis. The sugar determinations were carried out in Dr. W. Olmsted's metabolic laboratory in Barnes Hospital, and I wish to thank him for his assistance and Dr. Schwab for permission to use his cases.

These results were obtained by Shaffer's [2] method and at times also by Benedict's method for comparison. Sixteen such comparisons are shown in the table and in all cases, with one exception, the results were higher by Benedict's method. The average in our series is about 21 mg. per 100 c.c. higher reading by Benedict's method. As a rule, it is about 10 to 15 mg. per 100 c.c. higher than Shaffer's method. We have only four estimations of lethargic encephalitis in our series, and the average reading was 60 mg. per 100 c.c., by Shaffer's method.

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*From the Laboratory of Neurology, Washington University School of Medicine.

1. Foster, Harold E.: Hyperglycorachia in Epidemic Encephalitis, J. A. M. A. **76**:1300 (May 7) 1921.

2. Shaffer, P. A., and Hartmann, A. F.: The Iodometric Determination of Copper and Its Use in Sugar Analysis, J. Biochem. **45**: No. 2 (Jan.) 1921.

Findings in Various Neurologic Conditions

Name	Date, 1921	Diagnosis	Sugar Determination, Shaffer Method	Sugar Determination, Benedict Method	Blood Wassermann Reaction	Spinal Fluid Wassermann Reaction	Pandy	Cell Count	Colloidal Gold Test
1. F. F.	May 3	Combined sclerosis	53	—	Negative	Negative	++	18	2233210000
2. H. L.	April 26	Central nervous system, syphilis	50	60	++— — —	Negative	0	2	
3. C. McC.	May 14	Central nervous system, syphilis	67	—	Negative	Negative	—	—	
4. C. R.	May 14	Central nervous system, syphilis	160 138	106	—	++	—	—	Xanthochromia
5. L. S.	May 6	Central nervous system, syphilis	42	—	++++ +++ ++++ —	++++ — ++++ —	+++	125	
6. E. B.	May 3	Central nervous system, syphilis	50	62	Negative	++++ ++++ ++++ —	++	26	0122100000
7. J. W.	Feb. 7	General paralysis	60	73	++++ —	++++ —	+++	18	1155543310
8. J. W.	April 23	General paralysis	53	68	++++ — — —	++++ — — —	+++	13	5554443200
9. J. W.	May 5	General paralysis	69	83	—	++++ — — —	+++	11	5554321000
10. V. F.	April 27	General paralysis	45	52	Negative	+++ — — —	++	4	0012100000
11. H. M.	May 23	General paralysis	45	—	—	++++ ++++ ++++ ++++	+++	22	5554432100
12. M. M.	May 22	General paralysis	85	101					
13. M. M.	May 23	General paralysis	112	121	Negative	++++ — ++++ —			
14. M. M.	May 23	General paralysis	53	119	.....	.....	+++	26	
15. M. M.	May 24	General paralysis	77	143	.....	.....	+++	44	
16. A. K.	May 25	Tabes	72	83	Negative	.....	0	1	
17. E. T.	May 9	Tabes	69	71	++++ T —	Negative	+++	9	
18. M. H.	April 13	Tabes	64	—	Negative	++++ — ++++ —	+++	65	0001111000
19. A. B.	Feb. 7	Tabes	54	60	—	Negative	++	37	
20. F. J.	Feb. 16	Tabes	69	—	++	Negative	+++	92	
21. J. B.	May 24	Tabes	Trace	—					
22. J. R.	Jan. 25	Lethargic encephalitis	67	—	Negative	Negative	0	4	1110000000
23. H. L.	Feb.	Lethargic encephalitis	69	—	Negative	Negative	+	36	0021100000
24. J. M.	April 13	Lethargic encephalitis	62	—	Negative	Negative	++	1	1235543210
25. J. M.	May 26	Lethargic encephalitis	43	54	Negative	Negative	+	0	
26. C. M.	April 14	Psychosis	64	—	Negative	Negative	0	0	0000000000
27. H. P.	May 9	Psych.	74	—	Negative	Negative	0	0	0000000000
28. B.	Feb. 16	Hyst.	50	—	Negative	Negative	++	4	
29. G. B.	May 19	Neuros	72	—	Negative	Negative	0	0	
30. V. T.	May 2	Hyst.	50	—	Negative	Negative			
31. W. S.	May 14	Brain tumor	82	—	+	—	-		
32. D. McC.	May 21	Ps. B. palsy	56	106	Negative	Negative	.....	1	
33. R. T.	June 10	Lethargic encephalitis	64	—	Negative	Negative	0	1	
34. J. P.	June 13	Lethargic encephalitis	48	—	Negative	Negative	+	1	

These were all undoubted cases of the disease, and this unexpectedly low sugar content, determined by Shaffer's method, made us wonder whether our former results by Folin's method were not to be changed in their interpretation. Perhaps it is not sugar which is responsible for the higher value in encephalitis by Folin's method, but rather some other reducing substance or substances which may not interfere in Shaffer's method.

We have found it unnecessary to precipitate the protein before carrying out the sugar estimation in ordinary cases by Shaffer's method. In xanthochromic fluids and others with increased globulin the proteins should be precipitated as in blood sugar work. (See Case 4, C. R., in the table; 160 mg. before precipitation and 138 mg. after precipitation.) Case 31, W. S., was one of brain tumor which came to necropsy. The neoplasm, presumably a glioma, replaced part of the thalamus and lenticular nucleus of one side and was encroaching on the fourth ventricle. In Case 14, M. M., and in Case 15 the fluid was blood tinged, and the results, which are quite high, are not to be depended on; the creatinin, estimated by Benedict's method, was found to be 1.7 mg. per 100 c.c. of fluid in this case. In every case tabulated the fluid was sent to the laboratory immediately after puncture, and the sugar was estimated the same day or within twenty-four hours. We have allowed fluids to stand two or more days after puncture, and the estimation after this lapse of time was essentially the same as on immediate estimation. Most of the punctures were made about two hours after meals.[3]

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3. In addition to the references already given, the following will be found of interest:

Dopter, C.: Sugar in Cerebrospinal Fluid in Epidemic Encephalitis, Bull. de l'Acad. de méd. **83**:203 (March 2) 1920; abstr. J. A. M. A. **74**:1545 (May 29) 1920.

Weston, P. E.: Sugar Content of the Blood and Spinal Fluid of Insane Subjects, J. Med. Res. **35**:199, 1916.

Schloss, O. M., and Schroeder, L. S.: Nature and Quantitative Determination of the Reducing Substance in Normal and Pathologic Cerebrospinal Fluid, Am. J. Dis. Child. **11**:1, 1916.

Hopkins, A. H.: The Sugar Content of the Spinal Fluid in Meningitis and Other Diseases, Am. J. M. Sc. **150**:827, 1915.

Kraus, W. M., and Pardee, I. H.: Serology of Spinal Fluid and Blood in Epidemic Encephalitis, Arch. Neurol. & Psychiat. **5**:710 (June) 1921.

EPIDEMIC ENCEPHALITIS* (LETHARGIC ENCEPHALITIS)

ITS PSYCHOTIC MANIFESTATIONS WITH A REPORT OF TWENTY-THREE CASES

LESLIE B. HOHMAN, A.B., M.D.
Resident Psychiatrist, Johns Hopkins Hospital
BALTIMORE

The prime purpose of this communication is to analyze the psychotic features of epidemic encephalitis as they have been observed in a group of twenty-three cases admitted to the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital, between the spring of 1919 and the summer of 1920. It also seems worth while to report the cases in some detail and briefly to analyze them from other points of view, because they are so protean. In practically all instances these cases were referred to the clinic because of mental symptoms or behavior difficulties. It is, of course, not supposed that mental symptoms play as important a rôle in the disease picture as this group would indicate. The cases were selected for us, as it were, from the larger group which, for the most part, was admitted to the general hospital. On the other hand, this series does not constitute a small proportion of the general hospital group. Not more than 100 cases have been seen in the entire hospital, and in the psychiatric dispensary a number of cases were seen which could not be admitted to the hospital.

A study of the psychotic data has made diagnosis possible in some patients who presented few neurologic signs, and the mental picture has been found to offer valuable confirmatory evidence in doubtful cases with neurologic findings. Several characteristic symptoms have been made out as well as certain general reaction types. While the reaction types are not distinctive and decisive, they are often suggestive and helpful.

Nine syndromes are presented in the order of their diagnostic importance.

SYNDROMES IN EPIDEMIC ENCEPHALITIS

1 *Great Push of Talk Without Distortion of Stream of Talk and Without Mood Alteration.*—This was first noted in Case 14. When given the slightest opportunity, the patient poured forth a minute, detailed description of all of his thoughts and feelings. At first his talk was concerned with the material of his delirium and later with

* From The Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital.

discussion of the disease, its symptomatology, treatment, etc. The only way of stopping his talking was to keep every one out of earshot. After five months of the disease, this was the only symptom persisting. In Case 10, this was the only abnormality giving a clue to the diagnosis. The mind of the second patient was quite clear but he seemed to be unable to control the incessant flow of words. The manic type of association—flight of ideas, distractibility, etc., has not been present, nor does the mood show alterations to account for the talk. The patients are not distractible; it is difficult to divert the topic of conversation to other channels. Increase of general activity has been absent. Apparently all inhibitions to silence are off; the flood gates are down and some force seems to drive from behind. This symptom was present in Cases 5, 9, 10, 11, 12, 14, 20 and 21. Mention is made of this excess of talk by two other observers, Hall [1] and House.[2]

2. *Euphoria, Jocularity, Feeling of Well Being.*—This has been one of the most striking symptoms and has been noted by Wilson [3] and House.[2] The latter regards it as one of the most characteristic symptoms. Case 1: The patient said that she "felt like a new-born babe." The patient in Case 2 seemed quite jovial, said she "felt happy" and passed witty remarks about other patients, although complaining constantly about her condition. The patient in Case 3 said she "felt all right" although she asked constantly for medicine and was very restless and uneasy. The patient in Case 5 said he felt "very well" and was cheerful. The patients in Cases 6, 9, 10 and 11 described the mood as "good." The patient in Case 13 joked in a knowing way when his illness was mentioned. The patients in Cases 14 and 21 both "felt fine" though racked with severe pain. The patient in Case 20 was quite talkative and happy during the first week of the disease.

This sense of well-being reminds one in some respects of the euphoria of the paretic, but is less empty and vacuous.

3. *Alertness and Mental Clearness Immediately on Being Aroused.*—This is evidenced when the patient is once aroused, in spite of stupor and somnolence.

When the disease first began to be seen it was amazing how the patient dropped off to sleep during an examination. The clearness and alertness immediately on being aroused is equally surprising. One would not expect as much wide-awakeness in a normal person aroused from sleep. It is probably true that the somnolence appears deeper than it really is because of the frequent ptosis. The phenomenon has

1. Hall, A. J.: Epidemic Encephalitis, Brit. M. J. **2**:461, 1918.
2. House, W.: Epidemic (Lethargic) Encephalitis. Clinical Review of Cases in the Pacific Northwest, J. A. M. A. **74**:372 (Feb. 7) 1920.
3. Wilson, S. A. K.: Epidemic Encephalitis, Lancet **2**:7, 1918.

been observed in Cases 1, 2, 6, 7, 9, 15 and 23, which comprise about one half of all cases showing somnolence. Attention has been directed to this finding by Economo [4] and Wilson.[3]

4. *Delirium.*—Delirium has been noted in fourteen, possibly in fifteen, of our cases. This term means any psychosis in which there is a dreamy state with clouding of consciousness, disorientation and association in which drifting and groping are present. Following Hoch's [5] classification of the deliria as organic and psychogenic, it was found that practically all of our cases fell into the organic group. The content of the delirious utterances concerned almost exclusively topics which did not involve the personality of the patient in any of the instinctive realms. Occupation was the most common type seen (Cases 1, 5, 6, 9, 13, 14, 19 and 21). This has been the rule in cases reported by others. In three of our cases—14, 15 and 16, the delirium was accompanied by fear. In only one of these cases (14) was there any possibility of alcoholism. The only other case reported with fear, is one of Wilson's,[3] and it is doubtful whether the diagnosis of encephalitis was correct. Hallucinosis was present in seven cases, and it usually dealt with casual material—occupation objects, elevators, etc. In one case the patient saw animals with fear response.

5. *Stupor States.*—Five of our patients showed stupor at the beginning of the disease. These stupor reactions have been mistaken for schizophrenic reactions, but it seems to us that usually the differentiation is not difficult. The schizophrenic patient makes use of voluntary motor mechanisms and rarely is there the incoordinate tremor and tonic rigidity seen in the encephalitic. In addition, the stupor does not arise in a schizophrenic setting. Further, the clearness of the catatonic stupor is absent.

6. *Behavior Oddities.*—Impulsive or unusual acts totally out of keeping with the rest of the activity of the patient have been seen in seven cases. This is as one would expect in psychosis of organic basis. The impulsiveness of the paretic and of the arteriosclerotic patient is well known. Frequently, however, the oddity has been so unusual, unmotivated, and in some cases so silly, that it has been significant. For instance, the patient in Case 3 tried to commit suicide by hanging herself with a shoestring and without any real desire to kill herself. The patient in Case 1 got out of bed suddenly, dashed down the long corridor and, when brought back, said "Why do I do things like this?" She was not frightened. The act seemed quite unmotivated. Case 2:

4. Von Economo, C.: Die Encephalitis Lethargica, Leipzig and Vienna, 1918.

5. Hoch, August: The Problem of the Toxic-Infectious Psychoses, New York State M. J. **13**: (Feb.) 1913.

The patient bathed practically the entire day, wrote an extraordinary number of letters, and took huge doses of cathartics. Case 11: The patient talked in an odd, stilted way, and in a high-flown, silly manner. In the midst of a formal conversation he would swear violently without any anger. Case 13: Although the patient had been in bed for weeks and was very somnolent most of the time, he would get up suddenly, go to his wardrobe, pack his suit-case and announce that he was leaving. Case 15: This patient attempted suicide on two occasions by walking out of the third story window. Case 16: This patient had temper tantrums without precipitating causes and totally out of keeping with her former disposition. Case 17: The patient would get out of bed and wander about looking for medicine. She was not delirious and knew well that she was in isolation.

7. *Depression.*—This symptom has been observed by us in seven cases. In cases 2, 3 and 5, reported by Barker,[6] it was the most prominent symptom present. Flexner [7] and others [8] have called attention to this affective change. In our Cases 6, 7 and 21, it was transitory; but in Cases 15, 16, 18 and 20 it was pronounced and prolonged, dominating the clinical picture. Suicidal attempts have been frequent, and at least two patients were brought to the hospital for this reason. In Case 15, there was the formal alteration of thinking and activity which characterizes the manic depressive patient—that is, slowness of thinking and talking, especially in the morning. Case 17: This patient showed a great deal of puzzling bewilderment and fear, as well as depression. In Cases 18 and 20, there was no slowing.

8. *Emotional Instability and Irritability.*—These symptoms have been frequent, especially the latter. Tantrums and outbursts of anger have occurred without obvious cause. The lability and overflow of emotional response have suggested a pseudobulbar syndrome in Cases 1, 5, 8 and 9.

9. *Memory Defect.*—It was rather surprising to find so little evidence of real, permanent memory defect in our cases, since we are dealing with a disease in which there is demonstrable alteration of brain substance. In all four cases (8, 11, 13, 22) in which memory defect was demonstrated (of course the acute deliria are excluded), the prime difficulty was in recent and immediate (retention) memory. The

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6. Barker, L. F.; Cross, E. S., and Irwin, S. W.: Epidemic Acute and Subacute Nonsuppurative Inflammations of the Nervous System Prevalent in the United States in 1918-1919: Encephalitis; Encephalo-Myelitis; Polyneuritis; Meningo-Encephalo-Myeloneuritis, Am. J. Med. Sc. **159**:157 and 337, 1920.

7. Flexner, S.: Lethargic Encephalitis, J. A. M. A. **74**:865 (March 27) 1920.

8. Encephalitis Lethargica: A New Disease? J. A. M. A. **72**:414 (Feb. 8) 1919; also footnote 4.

patient in Case 8, in addition, confabulated freely. With an alcoholic history, this case might easily have passed as a Korsakoff syndrome. In fact, there is a similarity here to the alcoholic deliria which become chronic and pass into the Korsakoff psychosis. All four cases showed a preliminary period of delirium, with recent memory and retention difficulty. This type of memory difficulty, with a tendency to confabulate, is, according to Bonhoeffer, the characteristic feature of alcoholic delirium. It was pointed out in the foregoing that the patients can be easily aroused to take in their environment. This is also characteristic of the alcoholic. Fear appears in several of our cases. The protracted reductions of epidemic encephalitis have, then, several points in common with the chronic alcoholic deliria.

REACTION TYPES [9]

In our twenty-three cases, four reaction types can be made out: (1) depressive, (2) psychoneurotic-like, (3) delirious, and (4) organic. While these reactions have not been clear-cut and decisive, and all have some organic coloring, they present sufficiently clear reaction tendencies to deserve grouping, especially as the psychotic features may present difficult diagnostic problems.

The inquiry arises here, as in the psychoses of parenchymatous syphilis, "Why should one patient react with manic excitement, another with depression, and a third with a classical paretic picture?" Neuropathology offers no explanation. We believe that with diffuse organic change, the personality or constitution of the patient is apt to rupture at its point of greatest vulnerability. A personality or constitution able to maintain an adequate adjustment with the proper repression or sublimation of unsocial trends or of strong instinctive drives, breaks along these lines when the automaticity of adjustment is interfered with. Some of our cases support this notion. Case 15: This patient, petted and spoiled as a child, with a violent temper, and later humored by her husband, with a tremendous sex urge which her husband was unable to satisfy, reacted with temper tantrums, and openly made sex advances to her physicians. In addition she showed tremendous desire to punish and fondle her nurses and repeatedly asked her physicians to punish her for her bad behavior. Case 16: The patient, caught by the disease in the midst of a difficult personal situation (refusal of her teachers to allow her to attend a "hop," and the knowledge that her parents were unable to allow her to return to the same expensive school the following year), reacted with temper tantrums, depression and attempts at suicide.

9. Under the discussion of each reaction type a short psychiatric summary of the cases of that reaction are included. The detailed abstracts of the cases will be found at the conclusion of the paper.

Case 18: A man of worrying disposition, became depressed and attempted suicide when his health appeared to be gone. Case 4: This patient, who had a history of years of hypochondriasis, reacted with a "chronic invalid" picture.

1. *Depressive Reaction Type.*—This type of reaction was observed in seven cases but in only two were there formal thinking and activity alterations, that is, slowness of the manic-depressive reactions. The other cases resembled the reactive and psychoneurotic depressions. The depression was usually combined with delirium.

Case 6: Insomnia was observed at onset; later drowsiness and stupor. The patient felt sad and wished to die. He was alert when aroused; his spirits were good, although he was sad about being away from home. There were some date discrepancies but with spontaneous correction. He recovered in three weeks. (Midbrain, rubro-striate, bulbar, pyramidal involvement).

Case 15: The patient was a petted, spoiled child, violent tempered and highly sexed. The onset was acute, accompanied by somnolence; after three weeks, there was an onset of irritable nagging depression with two suicidal attempts. There were subjective loss of memory and confusion; people were talking about her. During the course of the psychosis she showed great sex tension, with sadistic and masochistic trends; she made frequent suicidal attempts. She recovered after eight months. (Midbrain).

Case 16: Onset occurred during a period of disappointment, with depression, insomnia, emotional tantrums and suicidal attempts. During the course of the disease she was agitated, restless, suicidal. There was subjective inability to think and understand; she was puzzled and fearful. She recovered in two and one-half months. (Midbrain and bilateral pyramidal signs).

Case 18: Drowsiness and stupor were present one week after onset. The patient had nocturnal delirium for three weeks, then became restless, sleepless, weak, excitable, exacting and depressed. She worried over financial difficulties and made two suicidal attempts. (Midbrain; cerebellar).

2. *Psychoneurotic-Like Reactions.*—Several of our patients were admitted to the hospital with a diagnosis of hysteria or functional disorder. Case 3: This patient took to her bed after a severe fright and was easily startled. She would scream in the middle of the night as if in terror, and appeared dull and out of contact when attempts were made to talk to her. (Midbrain, bulbar, rubrostriate and pyramidal involvement). Case 4: The patient became increasingly hypochondriacal during the course of her illness. She always complained of somatic symptoms and her organic signs were discounted by her family

physician. (Cerebellar?) Case 10: The patient had always been nervous, emotionally unstable and hypochondriacal. The encephalitic process increased the same symptoms. Case 17: This patient complained constantly of gastro-intestinal difficulty and headache. All his symptoms were easily removed by suggestion. Any kind of capsule gave him a comfortable day. (Cortical; midbrain; rubrostriate). Case 23: The patient complained of indescribable pain all over the body, constipation, pain in the stomach, and a host of other symptoms. Just how many of these complaints were due to organic changes seems impossible to say. Our attitude has been to treat them as psychoneurotic because this opened the door to the main therapeutic aid more than did the more organic hypothesis.

3. *The Delirious Reaction Type.*—This type has already been discussed under symptomatology.

Case 1: Emotional instability and sleeplessness were present; three days later, stupor and delirium; (disoriented, saw animals on the wall, behavior oddities). There was a feeling of well being; alertness was shown when aroused. (Midbrain; bulbar involvement).

Case 2: After three months of illness the patient became irritable, discontented, restless and antagonistic. She took large doses of cathartics, bathed excessively, wrote an extraordinary number of letters, and later said she was a queen and that a drinking glass was worth a million dollars. Still later, she was stuporous but alert when aroused; also delirious, talking about trips to the mountains and imaginary conversations with negroes. She complained about the treatment she received, but was cheerful and jovial. There was a gradual recovery after five months. (Midbrain involvement).

Case 7: Onset occurred with fatigability, weakness and periods of drowsiness. Later, he had a delusion in which he thought he was talking to his dead wife and sister; he had feelings of unreality. Delirium was combined with depression. The delirium became more profound with the fear state; he thought that he was being killed and that his food was poisoned, etc. Delirium deepened into stupor and death occurred, due to bronchopneumonia. (Bulbar).

Case 9: Occupation delirium was present from the onset. Later, the patient was somnolent but was easily aroused. There were periods of excitement during the delirium; the mood was "good." Recovery occurred after one month. (Midbrain; rubrostriate).

Case 12: The onset was accompanied by weakness, restlessness and pain all over the body. There was drifting talk with partial disorientation; the mood was "all right." Attention could not be held on arousal. Delirium was more pronounced with excitement and overactivity. Death occurred one month after onset. (Midbrain; cortical).

Case 14: The onset was acute with restlessness, tension and fear. He thought he was to be shot. He had occupation delirium with drifting associations, but he was oriented. There was an incessant flow of utterances. There was persistence of fear delirium with push of talk for several weeks; then subsidence of delirium occurred with residual of irritability and constant, incessant talking. Volubility was present after one year. (Midbrain).

Case 19: Onset occurred with sleeplessness and occupation delirium and irritability. Gradual recovery occurred after two months, with residual of alteration of the sleep curve and pains in the chest. Complete recovery took place after eight months. (Rubrostriate; bulbar; midbrain).

Case 20: The patient was talkative and happy during the first week; blue, inactive and quiet during the second week; he became sleepless. Depression then deepened; he complained of pain around the heart and smothering sensations. The latter resembled anxiety states and the patient felt as though he were dying and called for help. Then followed a period of muttering, fluctuating delirium. With the onset of profound rigidity the patient became mute and stuporous. After a period of many months he began to show that he understood and would answer by closing his eyelids. He gradually began to speak and gave evidence of clearness, but developed several paranoid trends—he thought that the intern had given him syphilis, cystitis, etc. He still shows profound rigidity after ten months. (Midbrain; rubrostriate; bulbar).

Case 21: Four days after the onset he complained of feeling nervous. He became restless and talked incessantly. He did not sleep from the onset until death seventeen days later. He moved about constantly as if in great pain, and his talk concerned his occupation. He was partially disoriented, and in spite of great pain said he was in good spirits. Attention drifted easily. (Midbrain; bulbar).

4. *Organic Reaction Type.*—This has been characterized by the presence of memory defect, mainly for recent events and retention. Good humor, a sense of well being and a tendency to confabulate were present.

Case 8: The patient had influenza February, 1919, followed by weakness and "nervousness." June, 1919, he had delirium; he improved, but had to give up work on account of mistakes. He felt confused and then became drowsy and delirious again. He began to confabulate; he wept and laughed with an overflow reaction. Reduction in memory and retention were present. Association assets were poor (Korsakoff syndrome). (Rubrostriate; midbrain; cerebellar; pyramidal involvement).

Case 11: Onset occurred with weakness and fatigue; he soon became irritable and for two weeks was speechless after a temper tantrum. His behavior was "peculiar"; he swore without anger or cause; his talk was stilted; his mind appeared to be clear but he did not know persons or places. Memory for recent and remote events was poor. There was a general reduction of association material. Speech and writing were careless. Symptoms persisted until death after one year. (Optic neuritis and pyramidal signs).

Case 13: Onset was gradual with loss of interest, fatigability and spells of unconsciousness. Memory showed gradual deterioration over a year. On admission to the hospital he was drowsy. He fell asleep during the examination but was easily aroused. He was disoriented; his mood was "good"; he wandered about the ward looking for imaginary persons and places; he thought detectives were hunting him; he was jocose and covered his mistakes by joking. There was a general reduction of association assets; he showed no insight. There was no change after eighteen months. (Bulbar; rubrostriate; cerebellar).

TABLE 1.—PSYCHOTIC MANIFESTATIONS

Case Number	Delirium	Feeling of Well Being	Alertness and Clearness When Aroused from Stupor	Over-talkativeness	Emotional Instability	Depression	Behavior Oddities	Memory Defect
1	+	+	+	0	+	0	+	0
2	+	+	+	0	0	0	+	0
3	0	+	0	0	0	0	+	0
4	0	0	Not somnolent	0	0	0	0	0
5	+	0	0	+	+	0	0	0
6	0	0	+	0	0	+	0	+ (?)
7	+	0	+	0	0	+	+	0
8	+	+	0	0	+	0	0	+
9	+	+	+	+	0	0	0	0
10	0	+	Not somnolent	+	0	0	0	0
11	+	+	Not somnolent	+ (?)	+	0	+	+
12	+	+	Not somnolent	+	0	0	0	0
13	+	+	0	0	0	0	+	+
14	+	+	Not somnolent	+	0	0	0	0
15	0	0	+	0	0	+	+	0 (?)
16	0	0	Unknown	0	0	+	+	0
17	0	0	0	0	0	0	0	0
18	+	0	0	0	0	+	0	0
19	+	0	Not somnolent	0	0	0	0	0
20	0	0	Not somnolent	+	0	+	0	0
21	+	0	Not somnolent	+	0	0	0	0
22	+	0	0	0	0	0	0	+
23	(?)	+	+	0	+ (?)	+	+	+

Case 22: July, 1919, the patient showed a let-down in his activity, endeavor and good spirits. January 1, 1920, he began to sleep poorly and was restless. January 14, he became drowsy and listless. During February he had "mild convulsions." At night he was slightly delirious. Toward the end of February he began to improve but continued to feel weak and fatigable, and memory was poor, especially for recent events. There was no change in his condition during treatment. (Cortical; midbrain).

The results in the foregoing cases are summarized in Table 1.

SUMMARY OF PSYCHOTIC MANIFESTATIONS

1. Nine symptoms have proved to be of frequent occurrence: (*a*) push of talk without distortion, (*b*) surprising alertness on arousal from stupor, (*c*) feeling of well-being and happiness, (*d*) behavior oddities out of keeping with general activity or mood, (*e*) emotional instability and irritability, (*f*) organic type of delirium at times accompanied by fear, (*g*) stupor states, (*h*) depression frequently the dominant mood, and (*i*) characteristic memory defect.

2. Four reaction types have been made out: (*a*) depressive, (*b*) psychoneurotic-like, (*c*) delirious, and (*d*) organic.

ANALYSIS OF CLINICAL COURSE AND SYMPTOMATOLOGY (SUMMARIZED IN TABLE 2)

1. *Onset and Signs of Infection.*—Onset: The onset was acute in thirteen cases, subacute in six and gradual in four. The most common symptoms were dizziness, pain in the head, neck, body and extremities

TABLE 2.—SYMPTOMATOLOGY

Case Number	Onset	Lethargy and Somnolence	Asthenia	Fever	Diplopia	Headache	Insomnia	Noise Hypersensitiveness	Pain
1	Acute	+	+	+	+	+	+	+	0
2	Acute	+	+	+ (?)	+	+	+	+	+
3	Subacute	+	0	+	+	+	+	0	0
4	Acute	0	?	+	0	+	+	0	0
5	Acute	+	+	+	+	+	0	0	0
6	Gradual	+	+	+	0	+	0	0	+
7	Gradual	+	+	+	0	+	+	0	+
8	Gradual	+	+	+	0	+	+	0	0
9	Acute	+	+	+	+	+	0	0	+
10	Acute	0	+	+	0	+	+	+	0
11	Subacute	0	+	+	0	0	0	0	0
12	Acute	0	+	+	+	0	0	0	+
13	Gradual	+	+	0	0	+	0	0	+
14	Subacute	0	+	+	+	0	+	+	0
15	Acute	+	+	+	+	0	+	0	0
16	Acute	+	0	+	+	0	+	0	0
17	Acute	0	+	+	+	+	+	0	0
18	Acute	+	+	+	+	+	+	+	0
19	Subacute	0	0	+	0	+	+	0	+
20	Subacute	0	+	+	+	0	+	0	+
21	Acute	0	0	+	+	+	+	0	+
22	Subacute	+	+	0	+	0	+	0	0
23	Acute	+	+	+	+	+	+	0	+

(probably root pain), headache, fatigability, excitability, restlessness, insomnia and acute respiratory disease symptoms. Nausea and vomiting, depression, staggering gait, muscle twitchings, etc., were occasional symptoms. Fever at the onset was mentioned in only three cases.

Fever has been present in all but three of our cases, and the history is doubtful in one of these three. It has usually been only slight or moderate (100-102.5 F.) except in fatal cases. There appears to be no correlation between the degree of pyrexia and the neurologic or psychotic symptoms. The fever has been irregular in all of our cases.

2. *Brain Infection.*—Headache was noted in seventeen cases. In some it was exceedingly severe and persistent.

3. *Disorder of Brain Stem.*—Diplopia was present in fifteen of the twenty-three cases. It was usually transient but persisted in a few for as long as one month.

4. *Involvement of Basal Ganglions.*—Lethargy or somnolence was present at some time in the course of the disease in fourteen cases; at onset in three, within the first week in five, and within three weeks in five cases. Somnolence was present for from a few days to many months, and frequently was intermittent. In two, it appeared late in the disease.

Insomnia was present in seventeen cases. This bears out the contention that the disease is incorrectly named. We have chosen to accept the suggestion of English observers that the disease be called epidemic encephalitis and the type specified when the etiologic factor is known. In many cases the insomnia was intense and prolonged. In some cases, with the coming of night the patient became wide awake and active. In one case (*22*), it was impossible to induce sleep.

5. *Cerebellar Involvement.*—Asthenia, more or less marked, has been a constant finding (eighteen cases). It may appear at any stage of the disease and usually persists for a long period after the disappearance of other symptoms.

6. *Middle Hindbrain Segment Involvement.*—Hypersensitiveness to noises was observed in five cases and was quite striking. A loud or shrill noise frequently caused intense suffering, and at times outbursts of anger and irritability.

7. *Spinal Root or Spinothalamic Mechanism Involvement.*—Pain: The pain was of various types. It ranged from general aching to severe lightning pains. It has been suggested that the lightning pains were due to inflammation of the spinal roots, although the absence of herpes casts doubt on this. It is possible that the spinothalamic mechanism was disturbed.

PROGNOSIS

Of the twenty-three patients in our series, five died. Three of them ran a chronic course (many months), and two died after an acute course (two weeks). Twelve have made practically complete recovery. Of the remaining six, two are in a critical condition, and the other four are on the road to recovery.

NEUROLOGIC FINDINGS

There was third nerve involvement in seventeen cases. Ten of these showed involvement of the extra-ocular muscles. Ptosis was

TABLE 3.—NEUROLOGIC FINDINGS

Case Number	Third Nerve					Fourth Nerve	Fifth Nerve	Sixth Nerve	Seventh Nerve		Ninth Nerve	Tenth Nerve	Kernig's Sign	Babinski Sign	Reflexes		Tremor	Parkinsonian-Like Syndrome	Muscular Twitchings	Nystagmus	Speech and Writing Defect
	Ptosis		Pupillary Changes		Extraocular Palsies																
	Right	Left	Right	Left					Right	Left					Deep	Superficial					
1	+	+	0	0	0	0	0	0	0	+	+?	0	0	0	0	0	+	0	0	0	+
2	0	+	0	+	+	+	0	0	0	+	0	0	0	0	+	0	0	0	0	0	0
3	+	?	0	0	+	0	+?	0	0	+	+	+	0	0	+	0	+	+	0	0	+
4	0	0	0	0	0	+?	+	0	0	0	0	0	0	0	0	0	+?	0	+	+	0
5	0	+	0	0	+	0	0?	0	0	+	0	0	0	0	0	0	0	0	+	0	0
6	+	+	0	0	+?	0	?	0	+	0	+?	0	0	+?	+	0	+	+	0	0	+
7	0	0	0	0	0	0	0	+	0	0	0?	0	0	0	0?	0	0	0	0	0	+
8	+	+	+	+	+	0	0	0	+	0	0	0	0	0	+	0	+	+	0	+	+
9	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	0	+	+	0	+	+
10	0	0	0	0	0	0	0	0	0	+	0	0	0	0	0	0	+	0	0	0	0
11	0	0	0	0	0	0	0	0	0	+	0	0	0	0	+	0	0	0	0	0	+
12	+	+	+	+	+?	0	0	0	+	+	0	0	0	0	0	0	+	0	+	+	+
13	0	0	0	0	0	0	0	0	+?	0	0	+	0	0	0	0	++	0	0	0	+
14	0	0	0	0	0?	0	0	+	0	0	+	0	0	0	+	0	+	0	0	0	0
15	0	0	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
16	+	+	0	0	+	0	0	+	+?	0	0	0	0	0	0	+?	++	0	0	0	0
17	+	+	+	0	+	0	0	0	+	0	0	+?	+	+?	+	0	++	+	0	+	0
18	0	0	+	+	0	0	+	0	0	0	0	0	0	0	0	0	+	0	+	+	0
19	0	0	+	+	0	0	+?	0	0	0	+	+	0	0	0	0	++	0	0	0	+
20	+	+	+	+	+	0	0	0	+	+	+	+	0	0	+	+	+	0	+	+	0
21	+	+	0	0	0	0	0	0	0	0	+	+	0	0	0	0	+	0	+	0	+
22	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	+	0	0	0	+
23	+	+	+	+	0	0	0	0	0	0	+?	0	0	0	0	0	0	0	0	0	+

present in thirteen cases. In three it was unilateral, in ten bilateral. Pupillary changes were noted in ten cases; unilateral in three. Eccentricity of pupils, reaction to light and not to accommodation and rigidity of pupils were the common form of alteration. An unusual feature of the pupillary changes was their variability and inconstancy.

The fourth nerve was involved in three cases, the sixth in four.

The fifth nerve was involved in five cases.

Seventh nerve: Thirteen cases showed facial weakness; weakness rather than paralysis was the rule. In only two cases was the seventh nerve involvement bilateral.

The ninth nerve was involved in eight cases, and the tenth in six, evidenced by difficulty in swallowing, difficulty in phonation, hiccup, etc.

The deep reflexes were altered in eight cases and the superficial in two. The usual change was inequality of the two sides. Babinski's sign was seen in only two cases, and in both the finding was equivocal.

Tremor was present in sixteen cases, usually coarse and irregular. In four cases it was marked. It involved, in various cases, practically the entire musculature of the body.

A parkinsonian-like syndrome with mask-like facies, propulsive gait, rigidity, etc., was present, in part, in five cases.

Hiccup was present in two cases. In one it persisted for over a year.

Areas of hyperesthesia were present in two cases (9 and 14).

Nystagmus, usually irregular, was seen in seven cases.

Speech and writing defects were noted in thirteen cases.

The usual abnormalities in writing were tremor and carelessness. The speech was usually thick, slurring and careless. Distortion was absent.

Peripheral neuritis, optic neuritis and masseter tenderness were each observed once.

Muscle twitchings were seen in six cases.

LABORATORY FINDINGS

The spinal fluid findings are shown in Table 4. Mention of pressure is omitted because in routine lumbar punctures we do not use a pressure gage. Also, so much depends on the position of the needle in the spinal canal.

The methods used for testing for globulin were those of Ross-Jones and Pandy. The globulin is reported as being from 1 to 6+, 6+ indicating the heaviest cloud that is ordinarily obtainable in a clear fluid, and 1 indicates a trace. The Wassermann test was made with three antigens and double and quadruple quantities of spinal fluid were

used with antigen (B) cholesterinized beef heart extract. In the colloidal gold curves we used gold solutions prepared according to the method of Miller, Fulton, Brush and Hammer.[10]

Cell Counts.—We regard over 10 cells per cubic millimeter as definitely pathologic and counts greater than 5 cells per cubic millimeter as suggestive of pathologic change. In all of our cases, except four,

TABLE 4.—SPINAL FLUID FINDINGS

Case Number	Duration of Disease	Character of Fluid	Cell Count	Wassermann Reaction*					Colloidal Gold Curve	Globulin Test	
				A	B	C	2	4		Ross-Jones	Pandy
1	8 days	Clear	110	0	0	0	0	0	1122100000	++	+++
	17 days	Clear	90	0	0	0	0	0	1112200000	++	++
	52 days	Clear	18	0	0	0	0	0	1222210000	++	++
2	4 months	Clear	10	0	0	0	0	0	1222100000	+	+
3	17 days	Clear	19	0	0	0	0	0	1233200000	(+)	(+)
4	2 or 3 months	Bloody	...	0	0	0	0	0			
	2½ or 3½ mo.	Clear	5	0	0	0	0	0	2322100000	+	+
5	55 days	Clear	38	0	0	0	0	0	4432210000	+++	+++
6	42 days	Clear	5	0	0	0	0	0	0222111000	++	++
7	5 months	Clear	21	0	0	0	0	0	1222100000	+	++
	5½ months	Clear	6	0	0	0	0	0	1233310000	++	++
8	6 months	Clear	0	0	0	0	0	2	5555555420	++++	+++++
	6 months	Clear	32	0	0	0	0	0	5555555421	++++++	+++++
	7 months	Clear	472	0	0	0	0	0	1444454210	+++++	+++++
	7 months	Clear	493	0	0	0	0	0	5555555220	+++++	+++++
	7 months	Clear	26	0	0	0	0	0	4444453210	++++++	++++++
	7½ months	Clear	43	...	...	...	...	...	0211244000	++++	++++
	10½ months	Clear	27	0	0	0	0	0	11123331100	+	++
9	46 days	Clear	14	0	0	0	0	0	1100000000	(+)	++
10	13 days	Clear	207	0	0	0	0	0	1111000000	+	+
	20 days	Clear	22	0	0	0	0	0	1111000000	+	+
	27 days	Clear	17	0	0	0	0	0	1112100000	+	+
11	9 months	Clear	34	0	0	0	0	0	1122210000	++	+++
12	15 days	Clear	31	0	0	0	0	0	1122211000	+	+
13	1½ years (?)	Clear	22	0	0	0	0	0	.....	+	+
14	18 days	Clear	30	0	0	0	0	0	1222100000	++	+++
	52 days	Clear	17	0	0	0	0	0	5522110000	++	+++
	89 days	Clear	12	0	0	0	0	0	3332110000	++	+++
15	4 months	Clear	3	0	0	0	0	0	1122100000	+	0
16	30 days	Clear	37	0	0	0	0	0	1110010000	+	++
17	39 days	Clear	?	0	0	0	0	0	1111100000	0	0
	43 days	Clear	?	0	0	0	0	0	1111100000	0	0
	50 days	Clear	48	0	0	0	0	0	1222200000	++	++
	60 days	Clear	?	0	0	0	0	0	1111000000	0	(+)
	70 days	Clear	?	0	0	0	0	0	1111222100	+++	++++
	113 days	Clear	15	0	0	0	0	0	2222100000	+	++
18	64 days	Clear	53	0	0	0	0	0	1211000000	++	++
19	60 days	Clear	21	0	0	0	0	0	1112210000	(+)	++
20	22 days	Clear	19	0	0	0	0	0	2322100000	++	++
	25 days	Clear	40	0	0	0	0	0	3443332111	++	++
	48 days	Clear	3	0	0	0	0	0	2231100000	+	+
	63 days	Clear	1	0	0	0	0	0	0111000000	+	+
21	6 days	Clear	16	0	0	0	0	0	0001110000	+	(+)
22	120 days	Clear	0	0	0	0	0	0	4433211100	++	++
23	150 days	Clear	6	0	0	0	0	0	2445221100	+	+

* A, B, C, refer to antigens; 2 signifies double and 4 quadruple quantities of spinal fluid used with antigen B.

there have been over 10 cells. The type was usually small mononuclears. Cell counts as high as 493 per cubic millimeter were encountered, with a usual increase of from 15 to 30. Of the four cases showing less than 10 cells, the patient in Case 4 was seen in the third

10. Miller, Fulton, Brush and Hammer: A Further Study of the Diagnostic Value of the Colloidal Gold Reaction, Together with a Method for the Preparation of the Reagent, Bull. Johns Hopkins Hosp. **26**: No. 298 (Dec.) 1915.

month of the disease; the patient in Case 22, who was seen in the fifth month, showed globulin increase and a modified paretic curve.

The study of the cell counts has shown such a uniform increase that we are loath to make the diagnosis of encephalitis without this finding. On the other hand, the cases reported from the early English epidemic did not show the cell increase, and it has been found from study of repeatedly taken specimens that the counts vary, and when the disease goes on to recovery the count is likely to fall, although not necessarily paralleling the course of improvement (Cases 1, 10, 14, 17 and 20). This fall in the cell count may explain the absence of cell increase in four cases.

Globulin Increase.—In this clinic any increase greater than a trace is regarded as suggestively pathologic. In every case but one, more than a trace of globulin was found.

Colloidal Gold Curve.—No distinctive type of colloidal gold curve has been discovered. Many types of curve have been seen—paretic, modified paretic and high and low syphilitic zone curves. So far no menigitic curves have appeared. The type of curve was not constant for any one case on repeated examination. Although the types of gold curve are not specific, we feel that a positive curve (no curve showing less than a 3 has been regarded as abnormal) offers valuable confirmatory evidence when present. Nine cases showed definite pathologic alterations.

Wassermann Reaction.—In only one case was there any fixation, namely, a slight fixation in quadruple quantities on one occasion in Case 8.

White Blood Cell Counts and Differential Counts.—Slight leukocytosis has been the rule in our cases (from 10,000 to 17,000). This increase has persisted in some cases for several months. The differential counts have shown no pathologic alterations.

CASE 1.—*History.*—M. K., aged 40, white, married, a housewife, was admitted to the hospital, Feb. 20, 1920, and discharged, April 9, 1920. She "always feels drowsy; dizzy at times; blurred vision; nervous." The family history was negative. Menopause began in December, 1919. Since that time she had been somewhat worried. She had been a cheerful, easy-going person, with an interest in people and a real fondness for them.

The onset of the present illness was sudden on Feb. 13, 1920. She awoke feeling dizzy and faint. Later, the same day, she complained of seeing double. She staggered when trying to walk and had difficulty in going up and down stairs.

During the week before admission, she had occasional headache and complained of general weakness. Vision became more and more blurred so that she was unable to read or write. At times she wept, at other times she laughed and sang on the slightest provocation. Once she became hilarious, said she felt fine, that everything would turn out all right and that nobody need worry

about her. Three days after onset the eyelids showed a tendency to droop, and this became more marked with the progression of the disease. Until the onset of ptosis she was sleepless. After that she was drowsy and frequently fell asleep while people were talking to her. Her drowsiness deepened into a stupor, but she could be aroused. She lost track of time; she imagined that she was in a hospital, and that her husband was at work (while he was at home); she thought that she saw animals, snakes, alligators, chickens and pigeons crawling on the wall. There was no fear connected with her hallucinosis. When aroused, she talked in a rambling fashion, drifting from topic to topic, and it was only with difficulty that her attention could be held. Throughout, however, when aroused, she was good-humored and said nothing was wrong with her.

The patient was very sensitive to noises and would awaken at times with a startled expression. Five days after onset she complained of difficulty and pain in swallowing, and saliva began to collect in her mouth. Her weakness continued and appeared more marked on the right side. Six days after onset herpes labialis appeared. Left facial paresis was noticed. At times she showed convulsive jerkings of the arms and legs. Her physician insisted that she had had no fever since onset.

Physical Status.—The temperature was 98.6 F., pulse rate 84 and respiration 20. The pupils were contracted and reacted to neither light nor accommodation.

The patient cooperated poorly, but apparently could not read. The eyegrounds were normal. There was marked bilateral ptosis; more marked on the right. There was no diplopia, nystagmus or strabismus. The nasolabial fold was obliterated on the left and there was weakness in closing the eyes and showing the teeth, but the forehead showed no weakness. There was impairment of sensation over the left half of the tongue which showed coarse tremor. The patient swallowed with difficulty and complained of peculiar feelings in the throat. No disturbance of sensibility was present. There was bilateral herpes labialis of the upper lip. The deep reflexes were active and equal. The Babinski and Oppenheim signs were negative. There was marked tremor of the hands and forearms in the extended position. There was some weakness of the right hand, and of the right arm and leg, more marked in the flexors. Finger-nose and heel-tibia tests were well performed, but the gait was wide and ataxic. The patient swayed in the Romberg position, but did not fall. She was drowsy during the entire examination. The blood pressure was: systolic 105, diastolic 60. The physical examination was essentially negative otherwise.

Blood Examination: The blood findings were: red blood cells, 4,880,000; hemoglobin, 78 per cent.; white blood cells, 10,880. Differential Count: Polymorphonuclears, 70 per cent.; polymorphonuclear basophils, none; polymorphonuclear eosinophils, 5 per cent.; lymphocytes, 22.5 per cent.; large mononuclears, 5 per cent., and transitionals, 2 per cent.

Spinal Fluid: The findings, February 21, on the eighth day of the disease, were: clear; slightly increased pressure; 100 cells; globulin, Ross-Jones test ++, Pandy, +++; Wassermann reaction, negative; colloidal gold curve, 1122100000. Urine: In the urine were 0.5 gm. of albumin per liter, occasional hyaline casts and a few white blood cells. Otherwise the urine was normal.

Mental Status on Admission.—The patient was stuporous but restless; arms and legs jerked frequently; at times her face had a startled, puzzled expression, and she then would be more alert and responsive. When aroused she

answered questions relevantly and directly, but tended to lose the thread of the conversation. She said she felt sleepy and described her symptoms; she "felt fine"—"like a new-born baby, only I'm so sleepy." She denied depression, ideas of reference, influence and persecution but admitted former hallucinosis She thought she was in another hospital, missed the date by a week and did not recognize the physician. Her memory showed slight date discrepancies but was, on the whole, quite good. Her speech showed hesitancy and slurring.

Course in Hospital.—Shortly after admission her temperature rose to 101 F. and fluctuated between 101 and normal for about a month; during the last two weeks it was normal. Her pulse was commensurate with the temperature. The respiration rate was normal.

Feb. 27, 1920: Stupor and drowsiness continued. At times she was disoriented and delirious, the delirium being occupational in character. Following lumbar puncture on February 21, speech became normal; the ptosis, left facial weakness and right hemiparesis disappeared. Diplopia recurred on February 23. On February 24 she suddenly jumped out of bed, ran through the corridor and when brought back to bed wept and said, "Why do I do these things"? Frequently she confused names of doctors and nurses. When aroused she was cheerful and euphoric.

March 5, 1920: During the second week in hospital all symptoms disappeared except the somnolence, which was distinctly less. She was correctly oriented and showed growing interest in the hospital activities. Lumbar puncture on the seventeenth day of disease showed a clear fluid, with 90 cells, and ++ globulin. The white blood cells numbered 9,240. She had some insomnia and received barbital in small doses.

April 9, 1920: Drowsiness had completely disappeared by March 18. She was discharged, well. There was no asthenia.

Case 2.—*History.*—N. C., a white woman, aged 63, divorced, a farmer, was admitted to the hospital, March 6, 1919, and discharged, April 12, 1919. Her father had a psychosis at 50. The type was unknown. Menstruation was accompanied by severe pain and occasionally by attacks of unconsciousness. She remained single until 50, when she married a man twenty-five years her junior. She divorced him after seven years.

During the summer of 1918 she was unnaturally irritable and unreasonable. In November, 1918, she had an attack of headache, nausea and vomiting and was in a stupor for one and a half days. She improved after two weeks in bed, but similar, though milder, attacks occurred at two to three week intervals until the end of January, 1919. The attacks are said to have been accompanied by fever. They started with flushing, fever and rigidity of the neck and shoulder muscles. The pain passed from the head to the rectum and then down the legs. On Jan. 29, 1919, the patient had another attack with vomiting. Two days later she was taken to a hospital. While in hospital she was discontented, irritable, restless and antagonistic to the physician. After twelve days she went home in spite of her weakness, and seemed more contented, although she showed some oddities—she took large quantities of cathartics, bathed practically the entire day and wrote an extraordinary number of letters.

Feb. 15, 1919, at midnight, she awoke with a scream and complained of the usual pain and stiffness in the head and shoulders. Three days later she again cried out saying some one had struck her on the left side of the head and "knocked her features off." The left eye was closed, and the left side of the face appeared "starched."

From February 18 to 25 she was quite restless, complaining constantly of symptoms of the head and said she had "an iron cross in her rectum." On one occasion she held a drinking glass in her hand, declared it was worth a million dollars and refused to allow any one to take it. She acted as if she were a queen, calling for her retainers etc. She was very sensitive to noises and screamed if the bed was moved, or if she was touched. From February 25 to March 3 she was stuporous and was brought to the clinic March 6, 1920.

Physical Status.—The patient was well nourished. The right pupil reacted to light and accommodation and was regular; the left was dilated and fixed. There was marked ptosis of the left lid with inability to move the eyeball up or down. The left eye was turned to the left; apparently third and fourth nerve involvement. Repeated examination showed no papilledema. The right nasolabial fold was flatter than the left. Facial movements were normal on both sides. Taste, deglutition, phonation and general sensibility were normal. There was no tremor of the hands, face or tongue. The biceps and triceps reflexes on the left were more active than on the right. All other deep reflexes were active and equal. The Babinski sign was negative. There was some stiffness of the neck muscles. Speech and writing were normal. The cardiovascular system was normal except for hypertension, the systolic blood pressure being 175 and the diastolic 115. Physical examination was essentially negative except for infected hemorrhoids. A roentgenogram of the head showed "areas of increased density in frontal region; sella normal." A roentgenogram of the sinuses was negative.

The urine was negative except for a moderate number of white blood cells. The spinal fluid was clear with 10 cells; globulin +; colloidal gold curve 1222100000. The Wassermann test was negative with three antigens in the blood and spinal fluid.

Mental Status on Admission.—The patient was drowsy and complained of being weak and cold. When aroused she was alert and at first answered questions distinctly and relevantly. She described her trip to the hospital but soon began to tell about wandering in the mountains and holding meetings with the negroes. She seemed quite jovial and said she felt happy and contented; she made witty remarks about the patients and seemed to understand the hospital routine, but she found fault with everything that was done for her. She fell asleep several times during the examination. She said her sister-in-law thought she was a princess and had tried to get possession of her property. She vaguely admitted having seen imaginary things.

She was disoriented as to place, said she was in another city and not a hospital, but she knew the date approximately and recognized the people around her. Her memory for remote events was fair, but poor for the recent past. She repeated seven digits correctly, but her general information was poor. She realized she was out of her head "by the things she said."

Course in Hospital.—During the first five days in the hospital the patient was delirious, complaining about everything that was done for her and demanding constant attention; she "heard her mother and sister outside the door," insisted on taking tub baths, and that she could not void. When an attempt was made to keep her in bed she struck at the nurse. Throughout this period, however, she was cheerful and good-humored in contrast to her previous period of complaints. At night she frequently rambled on in a confused fashion about negroes, etc. She forgot her meals and insisted that she had not breakfasted when she had just been served. Her complaints about nursing care and about headaches and pain in her neck and abdomen persisted until March 21. From

that time on she showed increasing interest in the ward activities and her friends. She was discharged April 12, 1919, practically recovered except for third and fourth nerve palsies.

Catamnestic Data.—The patient has been well up to the present writing, but there has been little change in the left eye; the third and fourth nerve palsies persist but have improved.

CASE 3.—*History.*—S. K., white, aged 23, married, a housewife, was admitted to the hospital, Nov. 8, 1919, and discharged Dec. 2, 1919. Her father died of brain fever (?) at the age of 28. She was friendly, sociable, and showed no signs of instability.

For two months preceding the onset of the present illness the patient had been run down and complained of indigestion. Three weeks before admission she had a bad cold and complained of severe headache and fatigue. Two weeks before admission she was frightened by a burglar and did not get out of bed again. She lay with eyes closed and did not speak. She was taken to a general hospital. There she lay with eyes partly closed, seemed indifferent to her surroundings, ate only when fed and voided only when taken to the toilet. At times she appeared apprehensive. There was some tremor of the fingers with general rigidity. Her voice was low and monotonous, and she obeyed commands in an automatic fashion. The rectal temperature was 100 F. She had 17,500 white blood cells per cubic millimeter, and a few days later 13,000. She became restless, insisted noisily on getting out of bed, and was admitted to the clinic, Nov. 8, 1919, with a diagnosis of hysteria and dementia praecox.

Physical Examination.—The patient was pale and cyanotic with a masklike expression. She was drooling and could not swallow. She lay with eyes closed. The general muscle tonus was increased, more so on the left. The head was retracted but could be flexed without pain. Kernig's sign was negative. She could raise her eyelids only part way. Ocular movements and optic disks were normal. The patient could open the mouth only slightly and could not move the jaw laterally. She could not wrinkle the forehead or show the teeth. She swallowed with difficulty. Her voice was low and monotonous, and there was distinct difficulty of articulation. The tongue could not be protruded beyond the teeth. There was no disturbance of sensibility. She complained of pain in the left arm and leg and refused fully to extend them, but no special tenderness could be made out. All deep reflexes were active, but those on the left side seemed to be more active. The superficial reflexes were active, there was no Babinski sign. The patient's gait was shuffling, with short steps. There was a coarse, bobbing tremor of the head with coarse tremor of the hands and jerking movements in the right arm.

The temperature was 100 F., pulse rate 76 and respiration 22. The remainder of the physical examination was negative. The white blood cells numbered 14,000 and included: polymorphonuclears, 82 per cent.; polymorphonuclear basophils, 1 per cent.; polymorphonuclear eosinophils 3.5 per cent.; small mononuclears, 12 per cent.; large mononuclears, none, and transitionals, 1.5 per cent. The spinal fluid was clear, with a trace of globulin and 19 cells; colloidal gold curve 1232200000. The culture was negative. The urine was negative except for acetonuria.

Mental Status on Admission.—The patient was restless, refused to stay in bed and wandered about the ward. She answered questions simply and to the point, and asked frequently, "Won't you give me some medicine to put me to

sleep"? She described her mood as "all right." She was correctly oriented, and her memory was good for recent and remote events. Retention, calculation and general information were adequate. She knew she was sick.

Course in Hospital.—The patient's restlessness persisted, and she could not be kept in bed. On November 9, she shouted and screamed and attempted suicide by tying a shoestring around her neck. Her temperature rose to 101 F., and her pulse rate was 140. She had to be fed through a tube because of dysphagia. Her general rigidity and tremor increased. On November 10, she could swallow food, restlessness decreased, and she was more cooperative. Right ptosis was marked, the tongue deviated to the right, and the left side of the mouth was less active than the right. On November 12, she complained of drowsiness and double vision, but the rigidity had disappeared. The temperature fell to normal on November 14, and remained so. Her behavior showed no abnormality except for tearfulness when she was not allowed to mingle with other patients. By November 22 behavior had become normal, and all neurologic signs had disappeared. She was discharged as well on December 2.

The diagnosis of hysteria had been made outside because of the great improvement of the ptosis and swallowing when the patient was emphatically urged. The diagnosis of dementia praecox was undoubtedly due to the haste with which any semblance of stupor is taken for catatonia.

CASE 4.—*History.*—F. W., white, aged 44, married, housewife, was admitted to the hospital, March 24, 1919, and discharged, Sept. 4, 1919. Her mother was a "chronic invalid" and had migraine. A maternal uncle had "mental trouble," and one brother committed suicide at the age of 19. The patient was a frail, irritable child. She began to imitate the invalidism of her mother and brother at an early age and became whimsical about her food. Menstruation was painful and accompanied by migraine. The patient had been hypochondriacal with innumerable complaints for years, had consulted many physicians and had had three pelvic operations without relief.

During the summer of 1918 there was an aggravation in symptoms, and the patient made some suicidal remarks. In January, 1919, she had an acute febrile illness for three days, in which her temperature was 102 F. Following this she was confined to bed and complained constantly of pain in the head.

In February she had an attack of tonsillitis (?), after which she felt she would fall forward; her jaw would drop, her hands and arms quivered when she raised them. On March 14, 1919, a jerking movement of the eyeballs was noticed, which increased on attempt at fixation. She began to fear blindness and insanity, and her hypochondriasis increased with constant demands for reassurance and attention. Since onset, she complained of headaches and vertigo.

Physical Status on Admission.—The patient looked sick and showed marked undernutrition. The temperature was normal. The glands at the angle of the jaw and the posterior cervicals were definitely enlarged.

The pupils were regular and reacted to light and accommodation. There was slight strabismus of the left eye; but the most striking feature was a most extraordinary lightning-like jerking of the eyeballs with rapid excursions in all directions, occurring spontaneously and lasting from one to two seconds. The otologist reported "rapid nystagmus, irregular in action and time." Lateral deviation checked it somewhat. Irrigation of the right or left ear with cold water caused marked vertigo and nausea after about one half the normal time. Oscillation increased after irrigation. Impression: marked over-irrita-

bility of the vestibular apparatus on both sides. Otherwise the physical examination was negative, except for some jerking movements of the muscles of the neck and a staggering gait. The Romberg sign was negative. Finger-nose test was well performed. The blood pressure was: systolic, 120; diastolic, 80. The eyegrounds showed no sclerosis. The white blood cells numbered 6,000 and the red blood cells 4,500,000; hemoglobin, 80 per cent. The urine was negative. Lumbar puncture, April 1, 1919, brought bloody fluid. Another on April 15 showed 5 cells. The globulin was normal; the Wassermann test was negative; colloidal gold curve 2322100000.

Mental Status on Admission.—The patient was correctly oriented, and her mental status showed no abnormality except persistent hypochondriasis—fear of insanity, dizziness, fear of blindness, pain all over the body, gastro-intestinal complaints, etc. Her judgment and insight were good.

Course in Hospital.—The patient began to improve shortly after admission. The nystagmoid movements, also observed during sleep, gradually decreased. On discharge the patient was practically well. The eye movements were observed only in an extreme lateral position. Vertigo was complained of only at rare intervals, and the hypochondriasis had disappeared almost completely, although at times the patient wanted reassurance.

The fundamental difference from any known type of nystagmus at first gave rise to the question of hysteria. But for hysteria it appeared too far beyond the possibility of imitation, and the accompanying twitches and rigidity of the neck and the entire attitude of the patient spoke against a purely psychogenic origin. Momentary eye tantrums without any conscious component except dizziness would describe the type better than the term nystagmus.

CASE 5.—*History.*—E. G. S., white, aged 27, married, a farmer, was admitted to the hospital, April 22, 1919, and discharged, June 11, 1919. His maternal grandmother was insane. His personal history was negative.

March 1, 1919, when he arose, the patient complained that everything was black before his eyes. On March 2, he felt "dazed" and dizzy; he saw double and staggered when he walked. He went to bed and for ten days was dull and talked incoherently about his farming. He could be aroused only with difficulty and complained of headache. During the third week of his illness he became restless, agitated, talkative and complained of unbearable headaches. He thought there were wheels in his head, and that his head was divided into several parts.

At the onset of the restless period his mouth became drawn to the right, and his eyes "rolled up and out"; there were twitchings on the left side of his face, and his whole body jerked. For the five weeks before admission there was difficulty in chewing, and weakness of the right arm. The patient became over-talkative, and was inclined to be obscene and to curse. The staggering gait continued.

Physical Status on Admission.—The patient was well nourished. There was ptosis of the left eyelid, and the pupils responded sluggishly to light but briskly to accommodation. There was no strabismus or nystagmus. The fundi were normal.

There was definite paresis of the left side of the face. No disturbance of deglutition or sensibility was present. The grip was weak and there was some weakness of the flexors of the arm on the right. Deep and superficial reflexes were equal and active. There was no Babinski sign and no tremor. The gait was reeling. The heel-knee and finger-nose tests were poorly performed.

There was swaying in the Romberg position and a tendency to fall backward. The blood pressure was: systolic 135, diastolic 90. The urine was normal. The blood Wassermann reaction was negative. The white blood cells numbered 10,300, which included polymorphonuclears 75 per cent., polymorphonuclear eosinophils 3 per cent., small mononuclears 17 per cent., large mononuclears 3 per cent., and transitionals 2 per cent. There was 78 per cent. hemoglobin. The spinal fluid was clear, with 38 cells; globulin +++. The Wassermann reaction was negative; colloidal gold curve 4432210000. The temperature was normal on admission. A roentgenogram of the head showed normal sella, but small dense shadows just above the temporal fossa.

Mental Status on Admission.—The patient was alert and cooperative; he showed a push of talk without distortion. He said he felt well and cheerful, but he wept when thinking of his friends. His memory was good, but he refused to cooperate in retention. He was correctly oriented and showed some insight into his condition.

Course in Hospital.—The patient's temperature fluctuated between normal and 101 F., until May 28, when it fell to normal and so remained. The pulse at times was irregular and rapid (130). The talkativeness and restlessness continued until June 5, 1919. He was cheerful and in a good humor always, but from May 2 to 11 he would burst into laughter or tears without adequate stimulus. He thought at times that his head was unclean and insisted on washing his hands frequently. He also complained of a bad odor in his room. For three days in the middle of May he was drowsy and sleepy. Facial weakness, ptosis and gait began to improve about the middle of May and progressed steadily to complete recovery, except for slight left facial weakness.

CASE 6.—*History.*—J. B., white, aged 46, married, a merchant, was admitted to the hospital, March 3, 1919, and discharged, March 27, 1919. His family history was negative. When 27 years of age, the patient had business reverses and was depressed and tearful for a short period but soon recovered. He was a hard worker, even-tempered, uncomplaining, but seclusive. His interests had been limited.

Six weeks before admission he had had a severe cold. Five weeks before admission he began to lose sleep and to be nervous. Three weeks ago he became drowsy and complained of pain in the back of the head and neck. Once he said he was sad, and that he would die. His drowsiness deepened into stupor, and for the week before admission he could not open his mouth or eyes or talk plainly and complained of "drawing" in his eyes. He also had some ringing in one ear.

Physical Examination on Admission.—The face was masklike, and there was marked rigidity of the entire body. There was ptosis of both lids, more marked on the right. The pupils reacted promptly to light and accommodation. Ophthalmoscopic examination was negative. There was some dysphagia and articulation of the labials was difficult. All deep reflexes were overactive and the superficial ones were easily elicited. There was slight tremor of the extended hands. The gait was spastic. The patient complained of pains in the jaws. The blood pressure was: systolic 142, diastolic 90. The white blood cells numbered 8,600; hemoglobin, 89 per cent. The blood Wassermann reaction was negative. The spinal fluid was clear, with increased pressure, 5 cells; globulin, ++; colloidal gold curve, 0222111000. The urine was negative. A roentgenogram of the head showed that the posterior clinoid was irregular and an area of increased density in the cerebellar region.

Mental Status on Admission.—The patient's actual alertness was in striking contrast to his appearance of stupor. He answered questions directly and relevantly and was interested in the ward activities. He said his spirits were good, although at times he was sad about being away from home. He was correctly oriented. His memory was good for recent events, and fairly good for remote events. He corrected his mistakes in dates. His retention was satisfactory. Calculation was excellent. He realized that he was sick mentally.

Course in Hospital.—The patient's temperature ranged between normal and 100 F. until the seventh day in the hospital, when it fell to normal and so remained. On the third day the face was drawn to the left—apparently a right-sided weakness. He complained of headache from time to time; the rigidity persisted. He maintained that he could not put out the tongue. On March 14, there was a positive Babinski sign on the right, and the reflexes were more active on the right. On March 16, the tongue deviated to the left, and there was impairment of taste over the right anterior part of the tongue. The ocular movements were quite sluggish. On March 27, 1919, the facial weakness had disappeared; the rigidity had also disappeared, although the reflexes were brisk. His mind was quite clear, and his memory showed no defects.

CASE 7. *History.*—J. T., white, aged 42, widower, a merchant, was admitted to the hospital, Nov. 29, 1919, and discharged, Dec. 26, 1919. His family history was negative. He was normal at birth and his development was normal. He was a successful boat captain. He was married, March, 1918, and his wife died in November of the same year. The patient apparently led a normal, happy life.

In November, 1918, the patient had influenza. July 1, 1919, he began to feel tired, weak and "achy," and had frequent periods of drowsiness, often falling asleep while at work or driving his automobile. Speech was thick, and he spoke in a low tone, at times in a whisper. At times he could not swallow. His symptoms increased in severity, and at times he was delirious; he imagined he was talking to his dead wife and sister, had feelings of unreality—he thought his legs were those of some one else, etc. He lost 60 pounds in weight. In November, 1919, he complained of pain across the back and legs. He is said to have had fever.

Physical Examination on Admission.—His temperature was 100 F., his pulse rate 90 and respiration 20. He showed obvious loss of weight. There was some tenderness in the sacro-iliac region and tenderness in the thigh and calf muscles. The pupils reacted to light and accommodation. There was no nystagmus or diplopia. Ophthalmoscopic examination was negative. The patient limped as he walked and used a wide base. He tried to shield the left leg.

His writing was tremulous and uncertain, and his speech was distinct but low in tone and monotonous. There was some thickening of the peripheral vessels. The blood pressure was: systolic 130, diastolic 60.

The urine showed a large quantity of albumin; otherwise it was normal; phenolsulphonephthalein test 50 per cent.; urea nitrogen of blood 25.5 mg. per 100 cubic centimeters of blood.

There were a left inguinal hernia and hydrocele. The red blood cells numbered 12,120, including: polymorphonuclears 71 per cent., polymorphonuclear eosinophils 3 per cent., lymphocytes 19 per cent., large mononuclears 5 per cent., transitionals 2 per cent.; hemoglobin 75 per cent. The spinal fluid was clear, under increased pressure, 21 cells, globulin ++. The Wassermann

reaction was negative; colloidal gold curve 1222100000. The roentgenogram showed slight infectious arthritis of the spine.

Mental Status on Admission.—Except for changing position frequently on account of pain, the patient showed no abnormality in behavior. His talk was relevant and direct. He said he felt "blue and downhearted" over his condition. He admitted the hallucinatory experiences noted in the foregoing, but insisted that it was dream material. He was correctly oriented, and his memory for recent and remote events was good. Insight into his illness was good.

Course in Hospital.—Five days after admission the patient became delirious, thought he was in a boat, that his food was poisoned, that men in his room were trying to kill him, etc. He imagined that he was in a heavy fog and could not get through it; he was restless, refused to stay in bed and voided on the floor. Delirium alternated with stupor until the time of his death.

His temperature fluctuated between 99 and 101 F. until three days before death, when it rose to 104 F. On December 23, there were signs of bronchopneumonia. The right eye deviated to the right. The urine showed heavy albumin and granular casts. The leukocytes numbered 18,920 on the 24th, the temperature was 104 F., respiration 44, and pulse 124. Blood cultures and spinal fluid cultures were persistently negative. On December 26, the patient died. Necropsy examination was refused.

Case 8.—*History.*—F. J. M., aged 53, a mail carrier, married, was admitted to the hospital, Sept. 25, 1919, and died, March 4, 1920. His family history was negative.

In February, 1919, the patient had influenza (?). He was in bed only one week, but afterward he felt weak and nervous, in spite of which he insisted on remaining at work until April, 1919, when he had a ten day attack of nausea, vomiting and headache. He became delirious and remained so until June, 1919, at which time he seemed well enough to return to work. After a few days he had to give up because he made so many mistakes in his work. Gradually he became more and more confused and increasingly drowsy and delirious.

Physical Examination on Admission.—The patient showed loss of weight and a temperature of 102 F. There was a gross, coarse tremor of the head and extremities. The tremor decreased with purposive movements. The left pupil was larger and oval. Both reacted slightly to light but well to accommodation. There was nystagmus on lateral deviation, right ptosis and weakness of the right side of the mouth. The tongue showed coarse tremor but no deviation. Sometimes backward, and at other times forward propulsion was noted. The deep and superficial reflexes were equal and active. There was swaying and falling in the Romberg position. Speech was slurring, careless and tremulous. Writing was tremulous and careless. The blood pressure was: systolic 158, diastolic 90. The urine was normal. The white blood cells numbered 8,800; hemoglobin, 95 per cent. The spinal fluid was clear, under increased pressure, contained no cells, globulin ++++. The Wassermann reaction was negative except for slight fixation in quadruple quantities; colloidal gold curve 5555555420. Spinal fluid culture was negative. A roentgenogram of the head showed the intracranial vessels to be markedly engorged and tortuous. There was an area of increased density above, and posterior to, the temporal fossa. The pineal gland was calcified.

Mental Status on Admission.—The patient lay quietly in bed and appeared to be in a good mood. He made occasional attempts to get out of bed. He

could not identify the direction of passive movements. When asked to carry out commands he merely repeated them, but did nothing. He said he felt mixed up. He responded slowly to questions and seemed to have difficulty in understanding them. He described his spirits as good, and said that he was happy, but he wept as he said it. He could give no reason for weeping. He was completely disoriented and when given an opportunity confabulated freely. His memory was poor for remote and recent events, and he showed perseveration in dates. His immediate retention was good (seven digits), but he forgot test phrases in a few minutes. His calculation and general information were poor.

Course in Hospital.—The patient's physical condition varied from time to time until death. The temperature ranged from subnormal to 103 F., usually from 99 to 101 F. Frequent blood and spinal fluid cultures were negative. He had been incontinent since admission. The neurologic status varied. The size of the pupils changed, and frequently the right would be larger than the left. Ptosis became more marked on the left. Occasionally ankle and patellar clonus could be obtained on the right, and the right deep reflexes were increased. The spinal fluid findings varied but always showed increase in cells and a paretic type of gold curve. Examination for tubercle bacilli gave a negative result. The Wassermann test remained permanently negative. His inanition increased, and large bed sores developed. He died March 4, 1920, from respiratory failure. Necropsy examination was refused.

The mental condition varied, but, except for occasional periods of brightness, showed no essential change. He constantly maintained that he felt fine, and that there was nothing wrong. He continued to confabulate freely. February 14, he stopped speaking and became stuporous.

CASE 9.—*History.*—T. W. S., white, aged 28, married, a crane engineer, was admitted to the hospital, March 2, 1920, and discharged, April 5, 1920. His family and personal history were negative.

Onset occurred Feb. 16, 1920, with pains in the back and face, especially over the forehead and jaw. The temperature was between 101 and 102 F. Four days later the antrum and frontal sinus on either side punctured and yielded pus (?). On February 23, tremor involving first the arms and then the head and body appeared. At the same time the patient became delirious. Three days later he complained of seeing double; there was paresis of the left internal rectus. The temperature was slightly elevated until admission, when it was 100 F.

Physical Status on Admission.—The temperature was 100.5 F., pulse rate 100 and respiration 20. The patient was well nourished, somnolent and weak. The pupils reacted promptly to accommodation but sluggishly to light. There were vertical and horizontal nystagmus and weakness of both eyelids. The eyegrounds were normal.

There was increased tonus in the sternocleidomastoids, and the trapezii seemed weak. The superficial and deep reflexes were normal. There was some hyperesthesia in the lower part of both arms. There was no Kernig's sign. There was coarse tremor on intention and occasional twitchings in the back and neck. The blood pressure was: systolic 135, diastolic 90. The urine was normal. The white blood cells numbered 16,360; hemoglobin 84 per cent. The blood Wassermann test was negative. Lumbar puncture yielded bloody fluid (fresh blood). Examination of the sinus showed no abnormality.

Mental Status.—The patient lay quietly in bed. He was somnolent but could be easily aroused, and would then answer questions. He tended to drift into talk about his occupation, at times growing excited about it, and

he gave the impression of attention difficulty. He described his mood as good. He was oriented for place, but not for time or person. His memory for remote and recent events was not accurate but showed no more distortion than the delirium would produce. Retention, calculation, general information and judgment were satisfactory when attention could be fixed. Speech and writing showed carelessness and slurring.

Course in Hospital.—On March 12, the patient's condition was practically the same as on admission, except that he was brighter. He was overproductive in his talk when started, but showed no especial distortion. He complained of pain in the back and shoulders. The patient began to improve rapidly, and by March 30 was almost well. The following abnormalities were, however, noted: asthenia, pain, soreness and weakness of the right arm, right pupil larger than the left, slight tremor of the arms and legs. The spinal fluid at this time was clear; it contained 14 cells; globulin +. The Wassermann reaction was negative; the colloidal gold curve 1100000000. The temperature remained normal after the eighteenth day in the hospital.

Case 10.—*History.*—L. B., white, aged 26, a widower, a huckster, was admitted to the hospital, March 20, 1920, and discharged, April 23, 1920. One uncle was insane. The patient has always exaggerated any physical complaint and was regarded by his family as nervous and unstable. October, 1918, the patient fractured the base of his skull. November, 1918, there was decompression, after signs of increased intracranial pressure had developed. Recovery was uneventful.

In March, 1920, the patient began to have a sense of pressure in the head with some bulging in the decompression wound. On March 14, he had a "dizzy spell" with vomiting. From March 4 to 17 he was unable to sleep, walked the floor, humming and singing and said he felt well but realized that he was sick. On March 18 he felt weak, restless and nervous, and there was some jerking of the limbs, head and eyes. Admitted to the surgical service with a temperature of 102 F., he was so restless and talkative that he was transferred the following day to the psychiatric service.

Physical Status on Admission.—The temperature was 101.6 F. Except for some slight weakness of the right lip and the old decompression scar, the physical examination was negative. The white blood cells numbered 16,900 on the day of admission and 10,600 on the following day. The patient had taken practically no fluid for four days before admission. Examination of the urine was negative. The spinal fluid was clear, containing 207 cells; globulin +. The Wassermann reaction was negative; colloidal gold curve 1111000000. The spinal fluid culture was negative.

Mental Status on Admission.—The patient was alert but somewhat restless. When allowed to talk, he showed overproductivity but no distortion. He described his mood as good, was correctly oriented and memory, retention, general information, judgment and insight were excellent.

Course in Hospital.—The patient slept well after the first night and showed rapid improvement in his mental symptoms. His talk became normal after a few days, and except for oversensitiveness to noise, and asthenia, he showed no abnormality. He left the hospital feeling well, although there was some asthenia. Lumbar puncture showed sudden decrease in cellular elements.

Catamnestic Data: Sept. 15, 1920, the patient was unable to sleep at night but was drowsy in the daytime.

CASE 11.—*History.*—E. W., white, aged 47, married, an engineer, was admitted to the hospital, Feb. 14, 1920, and discharged, Feb. 28, 1920. His family history was negative. His personal history was also negative. He used alcohol moderately.

On April 16, 1919, the patient returned from a business trip saying he was "all in" and that he had "fallen to pieces." He drove his motor car so recklessly that his family consulted a physician. He was put to bed for six weeks. During this time he is said to have had fever as high as 104 F. At the same time left-sided weakness, involving the face and extremities, was noted. He grew quite irritable and after a forced cold sponge bath was unable to speak for two weeks, after which his behavior had been peculiar, and he had had numerous hypochondriacal complaints. Since June, 1919, his condition had remained practically stationary.

Physical Examination.—The patient was poorly nourished and showed evidence of loss of weight. The pupils were normal, and there was no nystagmus or ptosis. Deviation of the eyes to the extreme right was maintained with difficulty. There was some optic neuritis on the right; physiologic cupping was absent, and there was some exudate alongside the vessels. The left fundus showed two hemorrhagic areas. The left corner of the mouth drooped. There was no sensory disturbance. The deep reflexes in the left leg were exaggerated. The Babinski sign was negative, and there was no clonus. The superficial reflexes were normal. There was some weakness of the left leg. The blood pressure was: systolic 140, diastolic 80. The urine showed a heavy cloud of albumin and hyaline and granular casts. There was no peripheral arteriosclerosis. Phenolsulphonephthalein was normal. The temperature was 100.5 F. Blood examination revealed: hemoglobin, 60 per cent.; red blood cells, 3,500,000; white blood cells, 11,000, including polymorphonuclears, 68 per cent.; large lymphocytes, 3 per cent.; small mononuclears, 20 per cent.; large mononuclears, 1 per cent.; transitionals, 8 per cent. The blood Wassermann reaction was negative. The spinal fluid was clear, with increased pressure, 34 cells, globulin + +, Ross-Jones, Pandy test + + +. The Wassermann reaction was negative; colloidal gold curve 1122210000. A roentgenogram of the head showed "intracranial vessels very much distended and tortuous. No evidence of any localized lesion."

Mental Status on Admission.—The patient was quiet and cooperative, but spoke in stilted, bizarre fashion. At times he enforced his remarks with outbursts of swearing. This was quite pointless; for example, "You would laugh if they would get me rid of this eye trouble, which keeps me from reading and seeing, and this stomach. I would call for my wife immediately to come for me. What am I here for? Damn! I haven't syphilis. Take your damn fool tests. I am glad to be through with them. Give me my stomach and my eyes and Hell! Good God! Do I speak to the point? (Any odd notions?) I am hesitating because I want to be damn sure that I am telling exactly what is true . . . absolutely none. No unpleasant feelings, no anything at all." The patient was partially disoriented, did not recognize the physician or know the date. His memory for recent events was poor, but fair for remote. His retention was only 5 digits. Calculation and general information were poor. His writing was careless and his speech showed some slurring. He had little insight. His mood he described as good.

Course in Hospital.—The patient's temperature varied between normal and 101 F. during his stay in the hospital. Neither neurologic nor mental status showed change. He was irritable at times and frequently confused and dis-

oriented. He lost his way about the ward and his talk remained prolix and full of circumlocution. He misused words constantly and lost the point of his talk, although it did not seem to be delirious in its meanderings. Provocative arsphenamin injections gave negative blood results. Two months after leaving the hospital the patient died. No details are known.

CASE 12.—*History.*—J. F. Y., white, aged 24, single, a clerk, was admitted to the hospital, March 5, 1920, and died, March 18, 1920. His family and personal history were negative.

The onset, which was acute, occurred Feb. 16, 1920, with neuralgia (?) of the ear. February 19 he quit work, became very weak and restless and was seized with severe pains all over the body (?) accompanied by generalized twitchings and tremor. He was treated for a "nervous breakdown."

Physical Examination on Admission.—The patient was well nourished. His temperature was 99 F., and pulse rate 104. He complained of "pains all through the body, twitchings and earache." The patient could not cooperate well, and apparently there was bilateral internal rectus weakness. There was marked horizontal nystagmus, with slow movements to the left, and bilateral ptosis. The fundi were normal. There was facial weakness on both sides, more marked on the right. There was no sensibility disturbance. Deep and superficial reflexes were equal and active. There was no clonus or Babinski sign. The abdominal muscles were held rigidly, and the abdominal reflexes could not be elicited. The Kernig sign was negative. There were occasional muscular twitchings in all of the extremities. Speech was slurring. There was coarse tremor of the extremities at times. The heart showed marked sinus arrhythmia. The blood pressure was: systolic 130, diastolic 90. There was marked pharyngitis. The urine contained numerous white blood cells and on culture gave a nonhemolytic streptococcus. Blood examination revealed 14,000 white blood cells; 80 per cent. hemoglobin; a negative Wassermann reaction; a negative blood culture; spinal fluid clear, with some increase in pressure; 31 cells; globulin +; the Wassermann reaction negative and a colloidal gold curve 1122211000.

Mental Status on Admission.—The patient was restless and tossed about as if in great discomfort. He appeared to keep contact only by effort; he answered questions readily but tended to drift off deliriously. He was disoriented for time and only approximately oriented for person. He described his mood as "all right." His memory was impaired but no more than the delirium would account for. Speech and writing showed slurring and difficulty in attention.

Course in Hospital.—The patient's temperature rose to 102 F. and remained elevated until his death, when it was 105. On the ninth day of the disease the pupils became rigid and a corneal ulcer appeared on the left. Severe cystitis developed and probably pyelitis. He became delirious and could not be kept in bed. At times he complained of seeing double.

On March 16, the patient began to fibrillate, and he died on March 18. The general tremor and abdominal rigidity persisted until the end. Necropsy examination was refused.

CASE 13.—*History.*—I. W. P., white, aged 40, married, was admitted to the hospital, Oct. 18, 1919, and discharged Dec. 2, 1919. His family and personal history were negative.

In the fall of 1918 the patient began to lose interest in his work and seemed tired and worn out. In September, 1918, he had severe temporal headaches accompanied by hiccup which recurred from time to time during the year.

He also complained of loss of hearing with tinnitus and had frequent dizzy spells. On two occasions during 1919 he fainted and was unconscious for half an hour. For the past year memory has been failing, but it has showed marked variations. At times he can remember well and at other times his mind seems blank. Since the middle of September, 1919, he had been very unsteady on his feet, and since the last week in September he had been incontinent.

Physical Examination on Admission.—The patient was sleepy and fell asleep during examination. The eyes showed no abnormality with the exception of some haziness of the disks. The tongue was protruded slightly to the right. There was marked tremor of the entire body, including the lips and face, which at times became violent. He was incontinent (?) whenever somnolent. His blood pressure was: systolic 120, diastolic 80. Spinal fluid findings were: increased pressure, 22 cells, globulin +, and negative Wassermann reaction. A roentgenogram of the head revealed no abnormality. The white blood cells numbered 8,200. (The differential count was normal.) There was slight prostatitis, moderate pyorrhea. Urine examination was negative.

Mental Status on Admission.—This varied markedly. When the patient was first seen he insisted on his wife leaving the room and said, "I feel as if I had spent a week in hell." He said he did not know how he felt, and then laughed. At times he was disoriented and failed to remember his examinations; he persistently wandered into the wrong rooms, and urinated in the tooth mug. He thought he heard the president of his company on the floor below. Then he lay quietly in bed and from time to time fell asleep. When aroused he smiled naturally but in a rather whimsical fashion. He answered questions for a time and then closed his eyes and refused to speak. He said his spirits were good, and his attitude suggested this. He admitted hallucinatory experiences but refused to discuss them. He thought there were detectives watching him, but there was no apprehension or distress about it. Orientation was only partial. He knew he was in a hospital but did not know the month or year. Retention was fair; he retained six digits. Calculation was poor. He joked about the examination to cover up his deficit. He showed no insight.

Course in Hospital.—His physical condition did not change during his stay in the hospital. From time to time he had periods of violent tremor which gave the impression of a chill. Hiccuping occurred from time to time and persisted for from 24 to 72 hours. His temperature was normal throughout his entire stay in the hospital. His mental condition varied markedly. He was never completely oriented, and his memory was persistently poor. Retention, calculation and insight showed no improvement. At times he was in fair contact with the environment—would talk about casual topics. He always joked when examined and tried to cover up his mistakes by bantering with the physician. At times he would pack his suit case and prepare to leave, but would not say when he was going. He was finally transferred to a state hospital near his home.

CASE 14.—*History.*—W. H. E., white, aged 24, single, a medical student, was admitted to the hospital, Feb. 2, 1920, and discharged, June 9, 1920. His father died at the age of 56 of an apoplectic stroke. His personal history was uneventful. He was shy, was diffident and moderately alcoholic.

During the fall of 1919 the patient was in better humor than he had been for years and friends remarked on his talkativeness. During January, 1920, the patient felt "off color," and his appetite was poor. Jan. 26, 1920, he had a severe headache and felt weak and tired. He gave up work on the 28th

when he discovered his temperature was 102 F. He was admitted to the medical service on January 30, complaining of headache and general malaise. On February 1, he developed sharp pains just above the left great trochanter; they ran down the thigh like an electric shock. During the night he became restless and tense. He told the nurse that "these men" were trying to shoot him and spoke of electrical connections which he had to fix. He was careless about exposing himself. On occasions he wept over imagined injuries and spoke constantly about his school work. He was oriented for place, time and person, but his talk was drifting.

Physical Status on Admission.—The patient complained of shooting pains down the outer side of the left thigh, which at times ran through the buttocks. He moved restlessly in bed and cried out as if in great pain. The pupils were normal. The patient complained of diplopia. Nystagmus and strabismus were not present. The eyegrounds were normal. His tongue protruded slightly to the left. There was an area of hyperesthesia over the outer side of the left thigh from the great trochanter to the knee and particular sensibility over the ischial tuberosity. The deep reflexes were sluggish and the left knee jerk less active than the right. Ankle reflexes were absent. There was no tremor or rigidity. The urine was clear. A blood culture was negative. The white blood cells numbered 12,800, including: polymorphonuclears, 72 per cent.; large mononuclears, 2 per cent.; transitionals, 4 per cent.; large lymphocytes, 2 per cent.; small lymphocytes, 2 per cent.; small mononuclears, 20 per cent. The pulse rate was between 110 and 120; temperature, 101 F.

Mental Status on Admission.—The patient talked constantly, describing his illness in great detail. Whereas normally he talked very slowly, his talk now was extremely rapid and continuous. He was well oriented, but his talk showed delirious drifting. It was only by reminding him of his subject that he could be persuaded not to describe his delirium as facts. He tended to describe imaginary injuries, and there was much fear accompanying the episodes. He recalled with a deal of feeling a rather frank medical discussion of his mental condition which he had heard at the bedside. He insisted that he felt fine were it not for the pain. His memory was quite good when attention could be held.

Course in Hospital.—The temperature remained elevated around 100 and 101 F. for two months. There was persistent tachycardia, but the electrocardiograms showed no abnormality.

The diagnosis of "flu" made at first was changed when the clear spinal fluid under increased pressure showed 30 cells (mononuclear), globulin + +, and a colloidal gold curve of 1222100000. On February 19 some weakness of the right external rectus was discovered, and also a tremor of the eyelids and legs. The number of white blood cells varied between 10,000 and 14,000. The diplopia was absent after the first month. The spinal fluid findings on March 19 were: a clear fluid, 17 cells, globulin + + and colloidal gold curve 3332110000. The blood and spinal fluid Wassermann reactions were at all times negative.

For a period of several weeks delirium was present, especially marked at night, and, while occupational for the most part, at times marked fear and terrifying experiences were observed. For instance, he thought that there were bandits about and he was to be killed. He refused to return to his bed on one occasion because it was occupied by a woman just delivered and everything was bloody. At times he confabulated freely. Once he said that he had been down town, and on another occasion that he had been home. He worried

about his school work and finances. Frequently all difficulties were solved in his delirium. He was at the horse races and won thousands of dollars. As the delirium began to pass, the patient became exacting and irritable. The slightest sound sent him into a rage, and he would relate endlessly all the discomfort of his stay in the hospital. At times he was extravagant in praise of the physician, and at others would tell at length how cases of his sort ought to be treated. Throughout the illness he was sleepless and obtained only two or three hours of sleep when given large doses of paraldehyd. Gradually the talkativeness and irritability decreased but on discharge, five months after admission, he was still overtalkative and would spend hours in discussing his illness with any one who would listen.

CASE 15.—*History.*—A. C. P., white, female, aged 29, married, was admitted to the hospital, April 20, 1920, and discharged Sept. 5, 1920. Her father was high-strung and nervous. A maternal aunt suffered from depression at the menopause. The patient was an only child, petted and spoiled by her parents, of a nervous temperament and worried over trifles. On several occasions she flew into a rage, but on the whole she controlled her temper. Her husband had difficulty in satisfying her sexually. She worried over possible pregnancy.

Feb. 1, 1920, the patient is said to have had the "flu" (?). At this time she complained of dizziness and could not focus. Following a short febrile period she did not gain strength and could not get enough sleep. Without warning she jumped from a third story window. Except for a broken heel, she sustained no injuries. The patient then admitted that she was despondent and worried and had tried to kill herself. The following day she complained of loss of memory and again jumped out of the third story window.

There were some signs of renal and visceral hemorrhage. A transfusion was performed, and she made a satisfactory recovery. She made several more suicidal attempts. Since the first fall the patient has complained insistently of loss of recent memory and retention of incidents, which she quizzed the nurses about. Morphia was required to produce sleep. Her behavior was capricious. She was easily irritated and would then pinch, slap, or bite whoever was near her. She remained markedly depressed and talked constantly of her trouble.

Physical Examination on Admission.—Physical examination was essentially negative except for evidence of the injuries and slight tachycardia. The blood pressure was: systolic 142, diastolic 88; the white blood cells numbered 5,600; the red blood cells 3,800,000; the hemoglobin content was 65 per cent. The spinal fluid findings were: two cells, no globulin, colloidal gold curve negative—1122100000. The blood and spinal fluid Wassermann reactions were negative.

Mental Status on Admission.—The patient lay quietly in bed but was irritable and complained about the precautions that were taken to guard her. She was well oriented. Her conversation was relevant, but there was also a lot of nagging about her treatment. She denied any suicidal attempts, was evasive about her illness, thought people were whispering about her, and said she felt blue and mixed up. Her memory showed no defect for remote events, and recent memory could not be adequately tested on account of her attitude.

Course in Hospital.—The patient's impulsive behavior continued. In the morning she was usually depressed and begged to be taken home. She made numerous crude attempts at suicide (eating a thermometer, falling out of bed, knocking her head against the bed, etc.). She confessed that her reason for

the first attempt at suicide was that her husband threatened to divorce her when he discovered some marital infidelity. She resented watching and pinched and choked her nurses. She admitted her impulsive behavior but said she could not control herself. She was under high sex tension and pleaded with the physicians to caress her; later she denied this in part. She was convinced that she was pregnant in spite of the negative results of examinations, and not until the administration of luetin tablets and the beginning of menses was she convinced. She was quite willing (one suspects too willing) to accept punishment for bad behavior. Memory and insight were excellent. She gradually improved and became less impulsive than on admission.

Sept. 15, 1920: The patient had recovered completely by Sept. 1, 1920.

CASE 16.—*History.*—M. C., white, aged 17, single, a student, was admitted to the hospital, April 28, 1920, and discharged, June 25, 1920. One cousin was insane. The patient was normal during childhood. Conscientious and hard working, she felt intensely about things but was not regarded as oversensitive.

On April 8, she was refused permission to attend a dance in a nearby university town. She was intensely disappointed over this, and when she also learned that her parents could not afford to allow her to return to school the following year, she wrote home that she was "utterly hopeless and miserable." A few days later she wrote that she was "sleepless, tired and melancholic" and "didn't want to live." From April 18 to 28 she had several emotional tantrums in which she wept and attempted suicide. She was excitable and felt that she was losing her mind; she insisted that she was a burden and wanted to die. On one occasion she thought she saw a lion in the room. Between outbursts she acted fairly naturally, but her outbursts occurred without apparent cause. On April 23 she saw double and on April 24 there was slight ptosis on the left. She was afebrile except for a temperature of 100 F. after a period of overactivity. The spinal fluid was clear, contained 22 cells and a slight amount of globulin. There was somnolence in the daytime but insomnia at night.

Physical Examination on Admission.—She complained of diplopia. Both external recti were slightly weak; slight right ptosis and some weakness of the right side of the mouth were present. There was a bilateral ankle clonus, but the Babinski sign was absent. The abdominal reflex was absent on the left. At times there was tremor of the entire body. She slept most of the day. The white blood cells numbered 12,680. The hemoglobin content was 85 per cent. The spinal fluid was clear; it contained 37 cells and increased globulin. The blood and spinal fluid Wassermann reactions were negative. The colloidal gold curve was 1110000000. Examination of the urine gave negative results.

Mental Status on Admission.—The patient was agitated and restless. She attempted to get to the window, apparently intending to throw herself out. She cried for her mother and seemed puzzled and fearful. She was correctly oriented. She said she could not think and did not understand anything. She repeated this again and again. She could not cooperate sufficiently to test memory, retention, etc.

Course in Hospital.—The suicidal attempts and agitation continued for several weeks, and then she became more cooperative. She appeared puzzled and easily frightened until two weeks before discharge. Her memory showed no defect, and gradually she resumed her normal activity. Except for a slight tremor, she was well on discharge.

CASE 17.—*History.*—T. C. H., white, aged 36, married, a miner, was admitted to the hospital, Jan. 7, 1920, and discharged, April 21, 1920. His family history was negative. His personal history was also negative except for congenital hair lip and associated speech disorder. The patient was illiterate.

The onset of the present illness occurred, Dec. 1, 1919, with frontal headache, which became progressively worse. December 15 severe pain developed in the back of the neck, which prevented sleep. Shortly before admission there was some visual disturbance, and his arms and hands became tremulous. He was dizzy and unsteady on the feet.

Physical Examination on Admission.—When first seen in the surgical service, January 7, he had amblyopia, anisocoria and left-sided ptosis. The same day he had two convulsions; his temperature rose to 104 F., the pulse was rapid, and he became comatose, restless and incontinent. The next day there was cyanosis and dyspnea, his temperature was 105 F., pulse 140 and respiration 36. Then developed incoordination of the bulbs, inequality of pupils, overactive reflexes, bilateral ankle clonus and the Babinski sign on the right. There was right facial weakness and marked rigidity of the whole body. Kernig's sign was positive. The fundi were normal. The blood pressure varied between: systolic 130, diastolic 104 and systolic 170, diastolic 112. Blood culture was negative. The red blood cells numbered 5,512,000, white blood cells 28,520, including: polymorphonuclears 82.6 per cent., small mononuclears 14.7 per cent., large mononuclears, 0.7 per cent., and transitionals, 2 per cent. The spinal fluid was normal; there was no film formation and no tubercle bacilli. On January 20 it showed 48 cells (mononuclear), globulin ++, colloidal gold curve 0111100000. The coma cleared after three days, and when the patient was admitted to the psychiatric service, January 30, the physical examination showed the same picture except for bilateral ptosis, right abducens weakness, exophoria of the left eye, jerking of the eyeballs, marked tremor of the legs, arms and hands, and a temperature of 100.6 F.; urine, negative.

Mental Status on Admission.—The patient's behavior was normal except for persistent complaint of constipation and headache, obviously hypochondriacal because both yielded to minute doses of bicarbonate of soda.

Course in Hospital.—The patient gradually improved and when discharged was normal, except for coarse tremor of the extended hands and the anisocoria. During the stay in the hospital the spinal fluid showed marked increase in globulin on February 10, with a colloidal gold curve of 1000222100, and on March 23, fifteen cells, + Ross-Jones test, Pandy, globulin, and a colloidal gold curve of 2222100000.

CASE 18.—*History.*—S. S. B., white, aged 39, married, a factory worker, was admitted to the hospital, May 2, 1920, and discharged, June 22, 1920. His family history was negative. He was a competent workman. Auto-eroticism had been present since the age of 12 until the present time; it was a source of much conflict. He was married at the age of 28, and had three healthy children. He was always inclined to worry, especially over finances.

The onset of the present illness was sudden on March 1, 1920, with dizziness and headaches; he then had blurred and double vision. After one week the patient became very drowsy, and he had to be washed and fed. From March 15 to April 6, usually at night, he thought he saw animals of all kinds, elevators going up and down with people getting off and on, etc. During this period he lost track of time and often did not know where he was or who

was with him. Since April 1 the patient had been sleepless, weak, nervous, excitable and despondent. His dizziness persisted, and he was unable to walk. He began to worry over finances and twice attempted suicide by turning on the gas.

Physical Examination on Admission.—There was slight anisocoria—the left pupil was larger than the right. There were rapid fatigability of the left external rectus, nystagmoid movement on turning the eyes to the right, tenderness over the masseters, some incoordination of the left leg as shown by heel-knee test, ataxic gait, swaying and falling in the Romberg position and coarse tremor of the extended hands. The spinal fluid was clear, containing 53 cells (mononuclear) and globulin ++. The Wassermann reaction was negative. The red blood cells numbered 4,820,000, the white blood cells 12,860, including: polymorphonuclear neutrophils 68 per cent., polymorphonuclear eosinophils 1 per cent., lymphocytes 24 per cent., large mononuclears 4 per cent., transitionals 3 per cent. The hemoglobin content was 78 per cent. The blood Wassermann reaction was negative. Urine examination was negative.

Mental Status on Admission.—The patient was restless, irritable and excitable, much disturbed and irritated by the slightest noise. He was correctly oriented. He said that he felt despondent and worried. His head felt "mixed up and not clear." Memory for recent and remote events was excellent. Retention and general information were good. He realized that he was physically and mentally sick.

Course in Hospital.—The temperature, which was 100 F. on admission, fluctuated between that point and normal until May 24, and then remained normal. It was with difficulty that the patient could be induced to leave his bed. He continued to be depressed, but gradually showed an increasing amount of activity. He was easily upset and went to bed on the slightest provocation. When refused constant attention, he threatened suicide. The slightest noise threw him into an irritable temper, and he would threaten to leave the hospital. When discharged, there was no evidence of any neurologic disorder except a fine tremor of the extended hands. The patient was transferred to a state hospital until recovery from the depression.

CASE 19.—*History.*—J. G., white, aged 38, married, a fireman, was admitted to the hospital, June 6, 1920, and discharged, July 28, 1920. One paternal uncle was insane. The patient had been an active, healthy boy with plenty of interests. He had no known sex difficulties. He had been a fireman for the past thirteen years.

During the first week of May, 1920, the patient had had a heavy cold with difficulty in breathing. He went to bed May 6, complaining of pain in the limbs, headache and nervousness. There was tremor of the legs. The patient slept little at first and began to talk wildly about fighting fires, pulled the furniture around to find the fire, saw moving pictures on the ceiling, etc. There had been marked irritability. At times the patient had difficulty in swallowing and there had been thickness of speech.

Physical Examination on Admission.—The pupils were equal and regular but eccentric. They were drawn upward and inward—the right more than the left. They reacted promptly to light and accommodation. There was no nystagmus or strabismus. The fundi were normal. There was a marked tremor of the lower jaw. Movements, however, were well performed. Speech was slurring and indistinct. The tongue protruded to the right. There was marked intention tremor of both hands. Deep and superficial reflexes were normal.

Examination of the urine was negative. The white blood cells numbered 6,800; the hemoglobin content was 92 per cent. The spinal fluid was clear, containing 22 cells (mononuclear) and globulin +. The Wassermann reaction was negative.

Mental Status on Admission.—The patient muttered to himself about fires. He was quiet most of the time but got up occasionally as if hunting for something. He was totally disoriented; he thought the physician was a lawyer and talked about being on trial for some street brawl. He spoke in a hazy fashion about being thrown out of bed and downstairs. His memory for remote events was good when his attention could be held. He showed some perseveration of words when asked to name objects, but his mind was so clouded that little stress could be laid on this. Recent memory and retention were poor. He wept during the examination, without apparent cause.

Course in Hospital.—The patient's mental condition gradually improved until by July 7 his mind was quite clear. Speech was still difficult and slightly stammering. There was some confusion of memory for his present illness, but otherwise he was quite clear.

Sept. 1, 1921: The patient complained of reversal of sleep curve and pains in the chest.

Dec. 28, 1921: The patient reported himself as entirely well.

CASE 20.—*History.*—C. A. B., white, aged 36, married, a laborer, was admitted to the hospital, March 30, 1920, and died, June 17, 1921. His family history was negative. He had broad interests, was moderately alcoholic, friendly and sociable.

March 12, 1920, the patient became dizzy. There were slight (?) fever and pains in the elbows. During the first week he was very talkative and happy but said nothing unusual. On March 19 he did not seem to have much life, was blue and kept to himself. On March 20, he began to tremble violently, although he was not cold. Sleep became poor, and he paced the floor at night. His depression became more pronounced. The attacks of tremor continued, and on the 29th his speech became thick and he had difficulty in swallowing. There was also diplopia.

Physical Examination on Admission.—The patient complained of "numbness in arms and legs, pain around the heart and smothering sensations." His temperature was 100 F. His pupils were regular—the left larger; reaction to light and accommodation was slight. There was lateral nystagums on looking to the left, but no strabismus. The eyegrounds were clear. Facial and jaw movements showed some weakness, but there was no inequality of the two sides. The patient was unable to swallow and speech was thick and weak. There was coarse tremor of the tongue, face and hands, and at times a coarser tremor involving the legs and arms. The deep reflexes were active but equal. The patient perspired profusely, pools of fluid collecting in the ears. The face was masklike. There was marked general weakness and some rigidity. The patient held positions when put in them. No disturbance of sensibility was present. Examination of the urine was negative. The white blood cells numbered 12,050. The spinal fluid was clear, containing 19 cells (mononuclear); globulin + +. The Wassermann reaction was negative in the blood and spinal fluid. The colloidal gold curve was 2322100000.

Mental Status.—The patient was correctly oriented, quite cooperative and interested in his examination, but depressed and anxious about his condition. He said that he felt as if he were dying and called out for help.

Course in Hospital.—The patient became more stuporous, the rigidity increased, and the tremor of the body became almost constant. On April 11 respiration became labored (32), and there was right facial weakness. His temperature remained around 100 F., the pulse reached 120. The Babinski and Kernig signs were negative. The urine showed some granular casts and albumin ++. The phenolsulphonephthalein was 50 per cent. in two hours. The white blood cells numbered 13,960.

At times he recognized those about him. His talk was muttering and indistinguishable, but chance words showed that there was some clouding.

On April 4, the patient's condition had improved slightly, but there was slight internal strabismus on the right and some left facial weakness. The following day the patient became comatose and incontinent and showed tremor of the eyeballs. The spinal fluid showed 40 cells and globulin ++, and a colloidal gold curve of 3443532111. His condition became slightly improved, and he again recognized those about him. His temperature remained about 101 F. The neurologic status showed no change. The leukocytosis on April 6 was 14,140. On April 12 there was a Kernig sign on the right side; knee and ankle reflexes were more active on the left. The upper abdominal reflexes were absent. The pupils were dilated and fixed. The rigidity was profound. The patient was, however, much more alert. He did not talk but obeyed commands and answered questions by opening and closing his eyes. On April 14, there was retention, and the patient had to be catheterized. The temperature was still elevated. On April 22 the general condition had improved, although fever, rigidity, tremor and the other neurologic signs persisted. By April 29 the patient was afebrile, but otherwise there was no change except left-sided ptosis. The white blood cells numbered 18,000. May 1 he developed pulmonary edema—respiration 48, pulse 146 and temperature 101.4 F. He was given strophanthin and epinephrin intramuscularly. The temperature rose to 103 F. He soon began to improve and by the morning of the 6th was in the same condition as before the pulmonary edema, but decubitus ulcers began to develop. On May 27, the patient spoke for the first time, saying, "It is about time to get well or die." At the same time he referred to some event that had happened a week before and tried to help put on his nightshirt. The rigidity and neurologic status continued. On May 15 the spinal fluid was normal.

On June 17, the patient's attitude was still fixed and rigid. He could perform simple, spoken commands. The pupils reacted briskly to light but poorly to accommodation. The left pupil was slightly larger and the eye movements jerky. There was tonic contraction of the jaw muscles; the head was slightly retracted and the neck stiff. The arms were adducted, semiflexed and pronated and the finger semiflexed with thumbs opposed and adducted. Full extension of the arms was restricted by contracture. There had been little change except that he seemed to take in what was going on around him. He said that his bedsore was less painful, and that the man in the next room was to be operated on (correct).

Case 21.—*History.*—H. G., white, aged 17, single, a boilermaker, was admitted to the hospital, May 21, 1920, and died, May 30, 1920. His family history was negative. He had had influenza, October, 1918.

May 13, he complained of pain at the vertex. The next day this extended down the sides of his neck, and later into the throat and ears. On May 17, he complained of feeling nervous. The same night he was restless, sleepless and talked incessantly. Since onset he had not slept and could not be put

to sleep even with morphia. He complained of blurring of vision and difficulty in swallowing. His talk gradually became incoherent and confused.

Physical Examination on Admission.—The patient complained of soreness of the head, neck, arm and epigastrium. The temperature was 101.2 F. The pupils and eye muscles were normal; there was no nystagmus. There was left ptosis. The speech was thick and careless and the voice hoarse and weak. There was no disturbance of sensibility. Deep and superficial reflexes were equal and active. There was marked tremor of the extended hands. The gait was unsteady and the Romberg sign positive. The urine showed acetone bodies and some albumin.

The white blood cells numbered 6,800; the hemoglobin content was 90 per cent. The spinal fluid was clear, containing 16 cells (mononuclear), globulin +, with a colloidal gold curve of 0001111000. The blood and spinal fluid Wassermann reactions were negative. The blood pressure was: systolic 180, diastolic 120. The heart was enlarged to the left.

Mental Status on Admission.—The patient was in constant motion, writhing about, sitting up, staggering about and complaining of great pain. He talked continually, calling his mother and sister, and constantly went through the movements of his occupation. When his attention could be held he answered relevantly, but his talk tended to drift off. Throughout he said that he was in good spirits, although crying out with pain. He was only partially oriented —he knew he was talking to the physician but thought he was at home. It was impossible to test memory, retention and calculation on account of poor attention.

Course in Hospital.—The patient's temperature remained at 102 F. until death. Although the eyes were closed most of the time, it was impossible to get him to sleep. On the 23d, he became more restless, complained of pain in the abdomen, hips and elbows, appeared stuporous and began to hiccup. On the 24th he had twitching of the abdominal muscles. On the 25th the delirium had become more pronounced, but his stupor also increased. The respiration became irregular, and the hiccuping increased. On the 26th bilateral ptosis was observed. He died on the 30th with respiratory failure.

Case 22.—*History.*—J. L. R., white, aged 63, a retired sea captain, was admitted to the hospital, April 15, 1920, and discharged, April 21, 1920. His family history was negative. He had general good health. He was good-natured, cheerful and social, but high-tempered. During July, 1919, there was a noticeable let-down in the patient's activity, endurance and spirits. In October, 1919, an automobile accident caused a brief period of unconsciousness, but no after-effects were noted except some nervousness and timidity.

In January, 1920, the patient began to sleep poorly and was very restless. January 14, he showed a sudden change, becoming drowsy and listless and he complained of seeing double. There was no improvement in February, and for ten days he had "mild convulsions" with twitchings, especially about the face and mouth. His tongue swelled (?), and speech was thick. At night he was mildly delirious. After the last of February he began to improve, but he felt very weak, was easily fatigued and his memory was poor, especially for recent events.

Physical Examination.—The patient complained of weakness and numbness in his right leg from his hip to his knee. The temperature was normal. The right pupil was larger and irregular; pupils reacted promptly to light and accommodation. There was no diplopia, nystagmus or strabismus. The eyegrounds were clear. There was no evidence of arteriosclerosis. There was

no weakness of the facial or jaw musculature. Speech was thick. No evidence of sensibility disturbance was present. Deep and superficial reflexes were equal and active. A slight tremor of the extended hands was present. The heart was not enlarged, and the peripheral vessels were not sclerosed. The blood pressure was systolic 118, diastolic 70. The urine was normal except for the presence of hyaline casts. Phenolsulphonephthalein excretion was 58 per cent. in two hours. The white blood cells numbered 7,500. The hemoglobin content was 75 per cent. The red blood cells numbered 4,032,000. A roentgenogram of the head was negative. The spinal fluid was clear, under no increase in pressure, and contained no cells; globulin ++; colloidal gold curve 4433211000. The Wassermann reaction on the blood and spinal fluid was negative.

Mental Status.—The patient was listless, moving little and then very slowly. His attention was good, but he was very solemn and did not smile. There was no spontaneous talking but he answered questions relevantly, promptly and with normal inflection. He said he was not depressed, but complained of some difficulty in thinking. Memory for remote events was slightly impaired; retention was poor. He could not remember a test address after five minutes and could repeat only five digits. He retold a test story, however, very well. Calculation was good. Writing was tremulous and uneven.

Course in Hospital.—During the patient's stay in the hospital for one week, there was no change in his condition. Repeated urine examination failed to show any more casts.

CASE 23.—*History.*—D. B., white, aged 38, married, a housewife, was admitted to the hospital, July 6, 1920, and discharged July 12, 1920. Her family history was negative. She had had headaches all her life. There had been progressive increase in weight since the birth of the first child. She had had a severe attack of influenza in the fall of 1918, since which time she had not felt as strong.

The last week in March, 1920, the patient had nocturnal earache and immediately afterward had an attack of generalized muscular twitching without loss of consciousness. Several more such attacks followed in the ensuing ten days. During the last week in April, bilateral ptosis developed with dimness of vision and diplopia. The patient improved gradually but complained a great deal of rectal tenesmus. While at another hospital, she is said to have had some fever and her systolic blood pressure was found to be 200. Preceding her admission to this hospital she had been sleepless, depressed, listless, and had had aches and pains in "stomach and limbs." For the week preceding admission, there was some difficulty in swallowing.

Physical Examination on Admission.—The patient was obese, with large pads of fat which had a doughy feel. The skin was coarse and dry, and the hair of the head and eyebrows was scanty. The temperature was 100.8 F. There was a bilateral ptosis, more marked on the left. The right pupil was slightly larger, both slightly ectopic, displaced upward and inward. Both reacted promptly to light but sluggishly to accommodation. No diplopia, nystagmus or strabismus was present. Both disks were clear, the veins injected, the arteries tortuous and a little thickened. There was no facial or jaw muscle weakness. Deep and superficial reflexes were active. There was no tremor. Gait, speech and writing were normal. Sensibility was not disturbed. The urine was clear. Blood pressure was: systolic 162, diastolic 110. The heart was not enlarged. The white blood vessels numbered 8,900. The hemoglobin

content was 95 per cent. The spinal fluid was clear, containing 6 cells (mononuclear), globulin +. The Wassermann reaction was negative, colloidal gold curve 2445221100. The blood Wassermann reaction was also negative.

Mental Status on Admission.—The patient lay in bed with eyes half closed, giving the impression of greater dulness than her talk indicated. Her spontaneous talk was concerned mainly with her somatic complaints, which were chiefly rectal pain and indigestion. She was not oriented correctly for date, but recognized place and person. She said she felt sad because she had to be sick, with five children at home. At times she was jocular and made rather poor jokes. Her memory for remote events was excellent, and, although she recalled isolated occurrences of the preceding twenty-four hours, she could not give a consecutive account. She was unable to repeat more than six digits. She showed good grasp in a test story. Her general information was fair, but the tests showed inattention. She realized that there was something wrong with her mind.

Course in Hospital.—The patient's condition remained practically unchanged during her six days' stay in the hospital. She was very hypochondriacal and at times restless and slightly confused. She would get out of bed to look for a cathartic after the nurse had told her she was bringing her one. On one occasion she thought the nurses were talking with other patients about her. She showed slight elevation of temperature throughout, up to 99 F.[11]

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11. In addition to the references given, the following may be of interest:
Bassoe, P.: The Delirious and the Meningoradicular Types of Epidemic Encephalitis, J. A. M. A. **74**:1009, 1920.
MacNalty, A. S.: Report of an Inquiry Into an Obscure Disease, Encephalitis Lethargica, Rept. Local Govt. Board on Pub. Health and Med. Subjects, N. S., No. 121, London, 1918, pp. 12-69; also Proc. Royal Soc. Med., London **12**: 1918 (1), Sect. Med. 1-23.
Maier, H. W.: Epidemic Encephalitis at Zurich, Schweiz. med. Wchnschr. **1**:249, 1920.
Tilney, F., and Riley, H. A.: Epidemic Encephalitis with a Consideration of Some of Its Prominent Clinical and Pathological Manifestations, Neurol. Bull. **2**:106-135, 1919.
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Abstracts from Current Literature

A STUDY OF NISSL'S STAEBCHENZELLEN IN THE CEREBRAL CORTEX OF GENERAL PARALYSIS, SENILE DEMENTIA, EPILEPSY, GLIOMA, TUBERCULOUS MENINGITIS AND DELIRIUM TREMENS. U. N. Noda, J. Nerv. & Ment. Dis. **53**:3 (March) 1921.

The author takes up the descriptive histology of the "staebchen" cell, reviews the literature and gives in detail his own studies on selected groups of cases. As to the morphology, the "staebchen" cell is a rod-shaped cell with a markedly elongated nucleus, the nucleus staining faintly and containing one or more nuclei. Threadlike substances going out from the poles are frequently the only evidences of protoplasm. The nuclei may vary a good deal as to shape and yet retain the elongated appearance. It is found in the cerebral cortex in various diseases and especially in general paralysis in which it constitutes the most important histologic finding.

The question of origin of this cell is discussed at length in a careful review of all the contributions on the subject. Nissl believed that they were found parallel with the blood vessels for the most part, but if numerous were likely to be irregular in distribution. Alzheimer substantiated the frequency of "staebchen" cells in paresis and the parallelism to the vessels in the cortex, but added that they were irregular in the deeper layers. He stated that they originated from glia. He held to the view of an adventitial origin as they were found around blood vessels in the adventitial spaces and resembled adventitial cells in so many ways. Cerletti advocated a glial origin but believed they were a part of an otherwise invisible vessel. Straeussler agreed with the view of a glial origin, claiming to have found glial fibers given off and present in large numbers wherever glia cells were numerous. Ris found these cells indistinguishable from adventitial cells of the vessel walls, though believing in a glial origin. DeRuck stated his belief in a fibroblastic origin. Mott wrote that they were "probably collapsed capillaries." Ranke advocated a glial origin. Dupre found them largely in the frontal and central convolutions in general paralysis, more numerous in the large pyramidal layers with a parallelism with the axis cylinders of the nerves, and thought they were due to the paretic condition. Persini believed that they originated from glia. Rosenthal affirmed an adventitial origin. Bonfiglio accepted both glial and adventitial origins. Rondini concluded that they were abnormal products resulting from a progressive process in the vessel wall. Ulrich found the greatest number in general paralysis, in the precentral convolution in the polymorphous layer, vertical to the surface, and with a definite relationship to the vessel alterations. In meningitis she found them in the molecular layer under the pia, stating that they are significant in the histopathologic diagnosis of general paralysis and cerebral syphilis, important in meningitis and multiple sclerosis, but in other diseases of little significance. She inclined to a mesodermal origin. Lotman found them in seven cases of glioma. Achucarro, in a study of rabies, found them in the stratum radiatum and explained that this condition was due to the structure of this region. He believed that they resulted from degenerative processes but might develop in part from ectoderm and in part from mesoderm. Sinchowicz stated that they were more frequent in those cases

of senile dementia which ran a rapid course and believed that they arose from glia. Bassoe and Hassin found them in epidemic encephalitis, especially near the infiltrated vessels in the pia and the cortex.

The author studied several groups of cases from the standpoint of prevalence and charateristics of the "staebchen" cells. He gives the histories and pathologic findings in detail. In the general paralysis group the general histopathologic picture given is that usually accepted and typical of the disease. He regarded all rod-shaped cells as "staebchen" cells and found them in large numbers, sometimes as transition forms from glia cells. They were particularly numerous in the frontal region and cornu ammonis, but fewer in the temporal and occipital lobes and cerebellum. In the white matter they were fewer than in the gray in the cerebrum, but in the cerebellum the reverse order obtained. In the cerebral cortex the greatest numbers were present in the middle laminae, especially in the small and large pyramidal layers—sometimes there were ten or more in a single microscopic field. The long axis of the "staebchen" cell in the cerebellum was vertical to the surface in the molecular layer, but parallel to the radiations in the marrow. In the cerebrum they were parallel with the radiations only in the deeper portions. Elsewhere they were irregularly placed. In the cortex the general direction was vertical and parallel with the long axis of the nerve cells, especially in the small and large pyramidal layers. No relation in the number of "staebchenzellen" and extent of nerve cell degeneration was found. Rod-shaped trabant cells were numerous wherever there was an increase in trabant cells. There was, however, a parallelism between the "staebchen" cells and glia proliferation. Various transitional stages between glia cells and "staebchen" cells and spider cells with elongated nuclei were found. "Staebchen" cells were found in colony-like formations. In regard to the relationship with blood vessels, there was a parallelism between the number of "staebchen" cells and the extent of perivascular infiltration. Sometimes elongated adventitial cells resembled "staebchen" cells (but were probably regressive capillary changes), and the author believes it may be the origin of some of them. Protoplasmic elongations from the poles were characteristic of these cells. The nuclei were from 15 to 30 microns in length. Pigment granules seen in some were of a brownish or yellowish color and situated mostly at the poles. Many were also found loaded with fat granules. The cells as a whole were well stained with toluidin blue, thionin and the Weigert stain. Many spider-like types of glia cells were seen with elongated nuclei, which the author believes should be called "staebchen-like" spider cells.

In the senile dementia group the usual arteriosclerotic changes were found generally. In addition, however, there was a swelling and splitting of the elastic lamina. Glia changes were both progressive and regressive. Some spider-like cells showed elongated nuclei. "Staebchen" cells were not numerous, perhaps two in each field, most common in the cornu ammonis, the white substance of the cerebellum and the molecular layer of the cerebrum. The direction of the cells was the same as in the general paralysis group, but the nuclei were shorter and there was less protoplasm than in paresis, and more frequently regressive changes were found. In the senile plaques the author found glia cells taking part, some having rod-like nuclei, but they were not definitely "staebchen" cells. There were some rodlike spider types of glia cells.

In the epilepsy group there was a chronic cell alteration, proliferation and regressive changes in the glial elements. "Staebchen" cells were few—one in a single field—with the greatest number in the cornu ammonis. In the delirium

tremens group there was chromatolysis, alteration and pigmentation of nerve cells. There was an increase in glia cells, some of them bearing fat pigment, as did the nerve cells. "Staebchen" cells were few in number, short in length, lacking in protoplasmic processes, the majority being loaded with fat. In tuberculous meningitis these cells were more numerous than in senile dementia but less than in general paralysis. The outer cortical layers and those places in which infiltration of the blood vessel walls was most pronounced contained the largest number of "staebchen" cells. They were also found in the pia. The morphologic characteristics were essentially the same as in the other diseases described, but the nuclei were shorter, and the protoplasmic processes were present. Some held pigment. In glioma the "staebchen" cells had more cytoplasm, wider nuclei and protoplasmic processes that were shorter than in general paralysis. They were more numerous in the tumor mass than elsewhere.

The author's conclusions are that "staebchen" cells are the most important histopathologic finding in general paralysis; that they appear in appreciable numbers in meningitis and are fewer in other diseases, with the exception of glioma. There are typical and transformation forms. He classifies them as: (1) those having elongated nuclei with protoplasmic processes, most frequently found in general paralysis; (2) those with short nuclei and scant protoplasmic processes, seen in glial proliferation and more common in senile dementia; (3) all curious and bizarre shapes; (4) transitional forms between "staebchen" cells and glia cells, occurring when glial proliferation is marked and including the "staebchen-like" spider (glia) cells. In general they parallel the surrounding elements and originate partly from ectodermic and partly from mesodermic tissue. In a discussion as to the theories of the causes of glia cells becoming "staebchen" cells, he considers a mechanical factor—rodlike forms produced by adaptation to available spaces, the results of osmotic pressure, and lastly the supposition that the special function which glia cells are called on to perform can best be accomplished in an elongated shape.

PATTEN, Philadelphia.

EPIDEMIC ENCEPHALITIS. K. BONHOEFFER, Deutsch. med. Wchnschr. **47**:229 (March 3) 1921.

Bonhoeffer sums up his experience with epidemic encephalitis as follows:

1. Prodromal Period: Headache is one of the first complaints. This is usually less intense than headache occurring in meningitis. It is most frequently localized in the posterior fossa and has a tendency to radiate to the neck and shoulders. Fatigue and dizziness are common. Temperature is usually present, but it is of low grade. Bronchitis or intestinal disturbances were encountered in only three of twenty-seven of his cases. Visual disturbances occur among the early symptoms in the majority of cases. Tinnitus and paresthesia in the distribution of the fifth and other nerves are not infrequent. The occurrence of anxiety and even delirium during the first few days is characteristic. A change of facial expression is usually noted at the onset. Insomnia or lethargy may occur. The prodromal period may last for several weeks.

2. Types: Bonhoeffer divides his cases into three main types: (*a*) lethargic; (b) parkinsonian, and (*c*) choreiform. He mentions the myoclonic forms but does not group them.

Among the ocular palsies, paralysis of accommodation is most common, next ptosis, and finally conjugate deviation. Bonhoeffer noted loss of light reflex or diminution to light and accommodation in a considerable number of cases. In a few cases paresis of the tongue occurred, with difficulty in swallowing. The sensory fifth nerve is seldom involved, but occasionally motor weakness is noted. Lethargy and insomnia are encountered rather constantly. Temperature is usually slight and of long duration. High continued temperature is usually looked on as indicative of complications.

Nearly all patients showed increase of globulin and a lymphocytosis in the spinal fluid. Hemiplegic symptoms of pyramidal tract character and cortical affections were not found in the author's series. The Babinski sign or ankle clonus was rarely noted; there was more frequently diminution or loss of the Achilles' reflex. Vasomotor disturbances, retention of urine and constipation were not infrequent.

These are the usual symptoms. Next in frequency is the choreo-athetoid type. Bonhoeffer does not believe in considering these two types as sharply divided from one another, but calls attention to the fact that both are extrapyramidal motor disturbances. There are cases, however, in which marked choreiform movements are found from the onset, and which from the pathologic findings must be considered as thalamic and subthalamic in character.

3. Diagnosis: The typical case offers no difficulty in diagnosis. Bonhoeffer states that with his increasing experience many of the old nondiagnosed cases have become clear. Thus, a case of acute paralysis agitans, as also a case resembling Huntington's disease which he saw several years ago, now seem clear to him.

4. Differential Diagnosis: Cerebral syphilis, general paralysis, catatonia, myasthenia and chorea must be considered in the long standing atypical cases. Syphilis at times is especially difficult to rule out, as Bonhoeffer has noted the occurrence of a positive spinal fluid Wassermann reaction in encephalitis. The value of stiff pupils has also been impaired by their occurrence in encephalitis. Chorea is at times difficult to diagnose as the more severe types are not uncommonly end stages of encephalitis.

Frequently the diagnosis can only be made because of the prevalence of encephalitis. The author mentions the case of a patient who complained of pain in the back of his neck with marked fatigue. Because of the epidemic, a spinal puncture was performed which showed increase of globulin and lymphocytosis, thus indicating the diagnosis.

A cerebellar syndrome is also recognized by the writer. In one case tremor, headache and delirium occurred; later marked static disturbances developed with nystagmus which gradually disappeared. The spinal fluid in this case also showed a lymphocytosis and increase of globulin.

Hiccup was observed in one case. Two patients had acute atrophy of the tongue, nuclear in type. The possibility of a localized acute poliomyelitis in the medulla was considered. Bonhoeffer does not believe that poliomyelitis and encephalitis have the same identity, as in the latter, nuclear lesions tend to recover.

Finally, there is another group which under the picture of a dissociation and delirium progress rapidly to death. In some of these cases one must satisfy himself with a diagnosis of acute delirium.

5. Course of Disease: The course of the disease is variable. The acute cases in which death occurs in a few days are rare. The illness usually lasts

from four to eight weeks or longer. The lethargy has continued for months in some cases. Marked variation in symptoms is not infrequent. Thus the rigid type may develop choreiform movements, the lethargic type, delirium, etc.

Patients with cranial nerve palsies show a distinct tendency to recover. Difficulty in accommodation is the symptom that has a tendency to remain longest. More obstinate are the disturbances of extrapyramidal motility. Pictures of Huntington's chorea and paralysis agitans have been observed to continue for more than a year. Bonhoeffer has also observed long continued psychotic manifestations, especially types resembling cocainism.

Paresthesias of the head and trunk, emotional weakness and hypochondriacal ideas are frequent residuals. Amnesic disturbances were not noted, and Bonhoeffer states that he has not seen them recorded.

Intellectual defect was not noted in any case. This is important as frequently the expressionless face makes one think it present.

6. Prognosis: The prognosis in Bonhoeffer's series does not correspond to that of the Vienna Clinic in which one third of the patients died. Bonhoeffer states that unless complicated by pneumonia or other troubles, the prognosis is not unfavorable. It is bad in cases of severe delirium or deep stupor with high temperature. This type, however, is rare as compared with the milder and chronic forms. As a rule, one may say that patients who survive the first few weeks will recover. Occasionally some complicating factor may develop, as in one of the author's cases in which paralysis of deglutition led to bronchial pneumonia and death.

The tendency to the development of pulmonary complications is well known. The majority of patients who came to necropsy died of pneumonia. The theory that these patients had some involvement of the dorsal vagus nucleus is worthy of consideration, but requires further anatomic proof. The tendency for relapses to occur is not unusual, but as a rule succeeding attacks are milder.

7. Pathology: It is the opinion in Bonhoeffer's clinic that encephalitis is a disease of fairly characteristic localization. Macroscopically, usually nothing is to be noted. In one case miliary hemorrhages occurred. Microscopically, a lymphocytic infiltration of the blood vessels occurs. Small hemorrhages and foci of active glia proliferation and cell destruction are also present. The gray matter of the pons is especially involved, but the cortex, cord and pia show infiltration. Large foci do not occur as a rule.

Restitution of destroyed areas is a characteristic finding. Judging by the general clinical and pathologic picture, there is little doubt that much of the damage is toxic in nature. At the present time it is impossible to separate the types of encephalitis from an anatomic standpoint.

8. Etiology: No etiologic factor is as yet known. Bonhoeffer was able in several cases to isolate almost in pure culture pleomorphous streptococci described by Economo, but the presence of this organism in other infectious processes causes the author to hesitate to consider it the etiologic factor. Inoculation of the spinal fluid and brain matter have proved of no value. The consensus of opinion appears to be, according to Bonhoeffer, that encephalitis is a localization of the "grip" infection in the brain.

9. Therapy: The author concludes his paper without offering any special therapeutic measures.

MOERSCH, Rochester, Minn.

SPEECH, ITS DEVELOPMENT AND INTEGRATION WITH THE INTELLIGENCE. MICHAEL OSNATO, M.D., Neurol. Bull. **3**:47 (Feb.) 1921.

Osnato's paper is a plausible attack on one of the bulwarks of clinical neurology—the diagrammatic conception of speech. He distinguishes sharply between articulate speech which is merely a kinetic act involving the whole body and speech in a broader sense which is an intelligent learned function dependent on the acquirement and use of sense symbols, sound, visual, gesture (kinesthetic and proprioceptive), olfactory, and tactile. Even in parrots and primates the ability to utter speech is correlated with other evidences of intelligence. Garner in his popular treatise describes nine monophrastic expressions, one phonetic sound, and at least thirty "words" used by the higher apes to express elemental emotions and communicate with each other. The level of this intelligence is in doubt. Kinnaman regards it as "intermediate abstraction" which is a more complicated form of mental process than the simple idea or notion or the mere memory of a particular sensory perception. The human animal develops intelligent speech through a series of evolutionary steps beginning with the baby or monkey or parrot stage and reaching its ultimate differentiated state which is the production and expression by means of spoken language of abstract thoughts and concepts. The latter acquirement is solely restricted to the human adult. The infant utters unintelligible sounds and learns by repetition of results or reactions in the parent to associate the sound with the fulfillment of a desire. For instance, "goo-goo" may be interpreted by the mother as a call for water and the infant then associates water and the satisfaction which this gives to its needs with the production of the sound which it has made. The pathways which determine speech are sound, visual, gesture (kinesthetic and proprioceptive), olfactory and tactile, and in some sense their exercise and development is individual and uneven and somewhat governed by environmental factors.

Turning to anatomopathology Osnato finds another weapon to use against the "brain pattern" idea. He says "the only association fiber pathways which have any definite degree of constancy and which can only be made out in the gross by anatomical dissection after hardening of the brain in appropriate fluids, are the cingulum, the fasiculi-longitudinalis inferiores and superiores and the arcuate fibers. Various other association bundles are known, but are inconstantly arranged and not always easily identified. Further, the author believes that clinicopathologic correlations are generally wanting and rather indicate that the adherents of the "brain-spot-aphasia" theory can only see the spot and not the remainder of the brain. He quotes Mautier (L'Aphasie de Broca, Paris, 1908, Vols. 1 and 2) to advantage. In fifty-seven cases in which the patients during life presented clinical evidence of aphasia, the foot of the third left frontal convolution was absolutely intact; in twenty-seven in which aphasia had been absent it was destroyed; and in only eight of 103 necropsy examinations was Broca's dictum borne out. Marie concludes that "there is not one case of tumor involving exclusively the foot of the left third frontal convolution in right-handed individuals which has ever caused aphasia. Many times this convolution has been completely destroyed by the tumor and no aphasia has resulted." Frankel and Onuf's statistics are also somewhat confirmatory (Frankel, J., and Onuf, B.: Corticale and subcorticale motorische Aphasie und deren Verhaltnisse zur Dysarthrie, Deutsch. Ztschr. F. Nervenheilk. **15**:312, 1899). Burchhardt's daring attempt to produce aphasia in two noisy, overproductive insane patients by brain operation is interesting. "He

removed 5 grams of the cortical gray from the foot of the first and second left temporal gyri and no word deafness resulted. Eight months later in the same patient he resected the top and foot of the left third frontal convolution and no agraphia or motor aphasia resulted. In another patient this surgeon removed a portion of the left supramarginal convolution and destroyed a considerable portion of Wernicke's sensory speech area and no aphasia resulted. In this last patient the left third frontal convolution was also resected. The patients continued just as voluble and full of noisy speech as before." Osnato asserts that "it is our belief that in a much greater number of patients the process of the acquirement of speech and intelligence has been so irregularly different and individualized that to talk of patterns and any longer to hold the diagrammatic conception of speech as a function localized in definite centers is impossible and unscientific and is in contradiction of all our anatomical and pathological data and our knowledge of the development of speech in children and articulate utterance in intelligent animals."

In general, Osnato's conclusions are not in excess of his premises. Several of the premises may be and have been less definitely interpreted. For instance, certain investigators would probably be unwilling to concede to primates much beyond mere memory for sensory perception. Again, additional clinicopathologic material might counterbalance the striking array which the author presents. Nevertheless, the conception of a pathologic brain spot as a sole determinant of clinical aphasia rests on a less sure foundation at present than it did in the past.

STRECKER, Philadelphia.

SENSORY APHASIA WITH REMARKS ON THE PATHOLOGY OF MEMORY. WALTER VON SCHUCKMANN, Monatschr. f. Psychiat. u. Neurol. **48**:232 (Nov.) 1920.

The patient, a man, aged 29, had a stroke in February, 1919, which resulted in a left-sided paresis. June, 1919, a second stroke was followed by loss of speech. The patient was right-handed. Eight days after the stroke, his wife noted that the patient did not understand what was said to him. He became irritable and violent and was admitted to the hospital, July, 1919. He died, December, 1919.

Necropsy revealed:

1. Atrophy of the posterior third of the first left temporal convolution and adjacent areas, reaching up to the angular gyrus.

2. A medullary cyst, the size of a plum, lying beneath the supramarginal gyrus.

3. Other slight pathologic changes which would seem to have no relation to the aphasia.

Clinically, the most striking feature was the patient's inability to express himself in speech. Spontaneous speech consisted of a mass of meaningless and confused syllables. In contrast to this, word-finding for concrete objects was relatively good. The results of his responses in the naming of concrete objects are:

	Percentage
Understanding of spoken words	90.0
Speaking from dictation	70.0
Spontaneous word finding	65.0
Reading aloud	22.2
Understanding of written words	60.0
Spontaneous writing	0.0
Copying	0.0

There is a striking difference between reading and writing. The percentage of reading can be explained on the basis of a sensory aphasia (first temporal convolution) plus some involvement of the angular gyrus. The complete loss of writing must have a different source. The author considers it a subcortical agraphia, due to the interruption of fibers from the writing center to the hand center by the subcortical cyst which lay beneath the supramarginal gyrus.

The author calls attention to the fact noted by Bastian and illustrated by this patient, that in partial lesions of speech centers, speech reactions are produced more easily by objects than by ideas. In this patient 65 per cent. of the attempts at word-finding for objects seen or felt succeeded, but word finding through association or ideation was nil. This the author tries to correlate with Liepmann's findings. Liepmann finds that the left hemisphere has a predominating influence in praxia; the left hemisphere alone can compass purposive movements without the object, whereas the right hemisphere is quite able to bring about purposive movements with objects. Similarly the author believes it possible that the right hemisphere through the right-sided sensory speech zone is able to produce a word-sound picture in the presence of the optic or tactile impressions, whereas only the left sensory speech zone can bring up the word sound picture through association or ideational paths.

The author then proceeds to a discussion of certain features of the pathology of memory, basing his remarks on the work of Semon. Semon speaks of the general characteristics shown by living organisms of being permanently impressed and changed by a single stimulus, as its engraphic receptivity. The permanent change produced as the engraphic effect of the stimulus is called an engram. The engram has a latent tendency to recur. The activation of this latent tendency he speaks of as ekphoria. An ekphoric effect on the engram may be produced by a return of the original stimulus or by a stimulus which has produced a related or associated engram. The ekphoric effect produced by the return of the original stimulus is greater than that produced by a merely associated stimulus (or stimulus producing an associated engram). The author points out that milder grades of disturbance of the engram will do away with its reproduction through associated stimuli, but will leave undamaged its ekphorability (ekphorierbärkeit) through a return of the original stimulus. This "hypesthesia" of the engram may explain certain features of aphasias. On recovery from sensory aphasia, speaking on dictation improves more rapidly than spontaneous speech. One generally explains this by saying that speaking on dictation is more resistant. A more plausible explanation is a partial disturbance of ekphorability. At the heights of the disease the ekphorability of the word sound engram is lost both for the original and for associated stimuli (anesthesia of the engram). As the disturbance clears, the ekphorability by means of the original stimulus returns, while that for associated stimuli remains lost. The original stimulus for word sound engrams is the heard word; and the test for the ekphoric effect of the original stimulus is repeating the word on dictation. Therefore, speaking on dictation returns while spontaneous speech remains disturbed. On the same basis the author attempts to explain the fact that in recovery from sensory aphasia understanding of spoken words recovers more quickly than speech. He then applies this same law to the case reported, explaining the slight involvement of understanding of spoken words in contrast to the marked disturbance of spontaneous speech by this same hypesthesia of the engram. This is offered as a possible substitute for his previous explanation, namely, the intervention of the right sensory speech field.

SELLING, Portland, Ore.

CLINICAL ENDOCRINOLOGY. WALTER TIMME, Neurol. Bull. **3**:3 (Jan.) 1921.

Timme introduces his comprehensive point of view of the internal secretory glandular system by dividing mankind endocrinologically speaking into three epochal life periods. The first, presided over by the thymus and pineal glands, embraces the span of years from birth to puberty (accretion, growth, and development); the second, under the domain of the gonads with control exerted both here and in the initial stage by the thyroid, pituitary and suprarenal glands, includes the period of life from puberty to prime (advance, aggression, and reproduction) and finally, gradual deterioration to dissolution (involution) marked by diminution in the activity of the last mentoined triad of glands.

The thymus is credited with the power of favoring somatic growth with an inhibitory influence on the sex glands. The clinical characteristics of thymic subinvolution, which is interpreted as an extension of glandular functioning beyond the time of ordinary involution are: "peaches and cream" complexion, absence of beard, mustache, and sparse or feminine type of pubic hair, bluish white teeth, large central and small lateral incisors with cutting edge instead of fang canines, flail-like joints, "torus palatinus" and delayed epiphysial-shaft union. In addition, there is "low blood pressure, slow pulse, hyperacidity, fatigability and usually subnormal temperature. The genital apparatus is usually small, but besides this it also shows some lack of differentiation, so that the penis emerges from a scrotal fold which may entirely surround its base." This seemingly clear picture is greatly obscured by the intrusion of the compensatory sympathicotonic mechanism of the suprarenals, pituitary and thyroid glands. There is in the classic thymic patient a psychic syndrome of childishness, imitativeness, self-centering, obstinacy, negativism, and in general an inability to bear the brunt of the struggle for existence. The converse of this, or precocious involution, produces contrasting physical and mental earmarks: somatic precocity in teeth, bones and hair; and psychically, initiative, easily aroused anger and resentfulness. There is "mature-precociousness" if one may use such an indefinite term.

Pineal subinvolution is supposed to determine retarded sexual development, "such retardation being of a direct growth character without the concomitant of reversion or lack of differentiation." There is also unusual muscular development. The reverse or premature pineal atrophy reveals radiographic "sand" before the twentieth year, indications of muscular insufficiency and relative genital and psychic overdevelopment.

There follows an interesting discussion of the thyroid-suprarenal-pituitary group. To quote the author, it is "this group that controls quantitatively and periodically the activity of the other internal secretory organs and the intensity, regularity and reactive capacity of the functoning of the vegetative nervous system. In short, it controls the automaticity of the individual in his adjustment to environment, in his reaction to fatigue, disease, to anger and other emotions, to competition in the struggle for existence, and prepares the organism physically to meet the shocks attendant on it." (One may fairly hesitate before this all-embracing claim.) In discussing the underactivity of the thyroid, nothing new is brought to light. Emphasis is placed on the numerous intermediate stages which may exist between frank cretinism or myxedema and normality. The anterior lobe of the pituitary gland often compensates for thyroid insufficiency. The understanding of certain of the more indefinite

hyperthyroid reactions becomes easier, if we can assume with Timme that the gonads and suprarenal cortex are directly asynergic with the thyroid and that on them depends the maintenance or loss of thyroid balance.

Because it rests on a more secure foundation it may be well to repeat the sympathicotonic syndrome of plus suprarenal functioning: (1) delayed peristalsis, (2) rapidity of pulse, (3) flushing of the skin, (4) lessening of the secretions generally, (5) mobile and frequently large pupils, (6) deficiency in the gastric juice and in its acidity, (7) heightened temperature, (8) a heightened blood pressure and a general increase of metabolism." Mentally we find a dynamic person with definite initiative, euphoric, optimistic and choleric. The deficient suprarenal patient is pictured as inadequate, lacking smooth-muscle tone, with a tendency to ptoses of the smooth-muscle viscera. Of pathognomonic import is the "Sargent line," when it can be made to appear on the chest in the immediate neighborhood of the thyroid gland (a white line obtained by stroking the skin and disappearing under epinephrin injection). Hyperadrenia may closely simulate hyperthyroidism, and it is important to differentiate between these two conditions. A patient with the former disease manifests a high blood pressure, less hyperhidrosis, a high red cell count, absence of gastric hyperacidity and a relative degree of bowel stasis.

The clinical cases of pituitary disease are said to be primarily due to a failure of compensatory effort of other ductless glands, notably the thyroid, suprarenal, gonads and pancreas. Timme's discussion of hyperpituitary and hypopituitary states, dyspituitarism and the radiography of the sella, though it presents nothing strikingly original, is well worth careful study.

The gonads are principally and pathologically disturbed through overactive and subinvoluted thymic changes and early pineal deterioraton.

The author's thesis closes with the presentation of an inheritance chart which traces the posterity of union between a giant and a diabetic. Of twenty descendants, there were two with dyspituitarism, one with hyperpituitarism, one with exophthalmic goiter, two giants and one osteomalacic dwarf.

Timme's comprehensive outline is timely. He is enthusiastic—at times perhaps overenthusiastic—but after all, his enthusiasm is founded on original investigation, the research of other students and on clinical experience. Perhaps he is permitting himself too much latitude in the interpretation of certain sets of facts, but after all, inference and speculation, which is the forerunner of more profound knowledge, cannot be too narrowly restricted. In other words, there is permissible and indeed necessary for the formative stage, a certain amount of what has been called scientific guesswork. The certainty that future studies will result in more or less radical modification of the present point of view is recognized clearly by the author. Too much apology for the existing conception of the rôle of the endocrines in neuropathology is out of place. In the first place there are at least several indisputably established disease conditions which rest on as firm a foundation as do typhoid fever or tuberculosis. They provide a scientific basis for more extended investigation. Furthermore, the internal secretory organs lend themselves to accurate physiologic and biochemical study. Surely the field which is being so industriously tilled will yield a rich diagnostic and therapeutic harvest!

STRECKER, Philadelphia.

THE MENDELIAN LAW AS APPLIED TO THE PSYCHOSES, WITH SPECIAL REFERENCE TO SCHIZOPHRENIA. E. BLEULER, Schweizer Arch. f. Neurol. u. Psychiat. **1**:19-40, 1917.

Rudin has finally brought his investigation of the inheritance of the psychoses according to the Mendelian law to a conclusion. He found that of 701 families, including 4,823 individuals, 15.86 per cent. had schizophrenia, and that there were other types of psychosis in 1.63 per cent.; all told, 17.49 per cent. suffered from some form of psychosis, making allowance for those who were too young and might develop symptoms later, and those above the age of 40, when the development of schizophrenia is no longer expected. He found that 4.48 per cent. of persons with practically normal parents suffered from this illness. According to these figures, it is apparent that schizophrenia cannot be an expression of mendelism.

As the younger siblings developed schizophrenia more often than the older ones, it seemed to Rudin that other factors must be present in the production of the disease. It was difficult to determine from Rudin's work exactly what factors were inherited and what psychic manifestations could be considered as hereditary equivalents. It has, moreover, been noted in schizophrenic families that other types of psychoses may appear in the parents; thus, in thirty-five the type was organic, in sixty-five functional, in ten unknown, and in seven alcoholic. Only about one third of the mentally diseased parents of schizophrenic children had the same type of psychosis; and another third had functional disorders, of which about one-third were manic-depressive. While schizophrenic persons frequently have manic-depressive parents, Rudin was unable to find any instance of manic-depressive descendants of a schizophrenic ancestor.

Bleuler criticizes Rudin in so far as the latter selected schizophrenia and dementia praecox for his studies, believing that the disorders could be diagnosed with the greatest certainty. In Bleuler's opinion dementia praecox is of all psychoses, perhaps, the most difficult to be certain of. At the present time, classification of the various subgroups of schizophrenia present almost insurmountable difficulties. Bleuler is convinced that the conception of schizophrenia includes a number of entirely different types of diseases.

Just exactly what should be inherited in these types would be difficult to define. Often nothing is apparent of the disease until the illness has become well developed. We must be able to recognize what he describes as hereditary schizophrenia in contradistinction to manifest schizophrenia; these are related to each other by complex etiologic factors.

To be of definite value an investigation into the heredity of schizophrenia must take the following points into consideration:

1. The conception of dementia praecox, including the hereditary psychosis as well as the clinically manifest psychosis, must be well defined. It must either be uniform in its clinical appearance, or if polymorphic the various manifestations must be well understood.

2. The causal relations between the hereditary psychosis and its manifestations must be known.

3. The diagnosis of the disease must be certain in practically all cases.

4. Those who are the descendants of a homozygote must be manifest schizophrenics for it must be possible to distinguish the dominant and recessive homozygote from the heterozygote.

5. Conditions must permit that all patients in the investigated family can be examined.

6. Heredity must not be limited to either sex.

7. The mortality of the R R-, R D and D D types must be about the same.

8. There may be no shifting of the dominant.

9. No secondary factor may change the figures in any substantial manner.

10. The disease must not appear except through inheritance.

Of these considerations, one to five are completely unknown at the present time; the remaining points are for the most part not well understood. Bleuler thinks also that it is important to investigate not only the families of schizophrenics, but also those of all other types whether psychotic or normal. The relation to alcoholism, manic-depressive insanity and epilepsy must be well understood. The apparent relationship of schizophrenia and alcoholism suggests a polyhybrid genesis of the disease; indeed, the possibility suggests itself that to the schizophrenic gamete must be added some other factors needed to make the disease manifest, in this way really producing an equivalent hybrid.

WOLTMAN, Rochester, Minn.

ANEMIA IN OLD AGE. H. CURSCHMANN, München. med. Wchnschr. **68**: 172 (Feb. 11) 1921.

According to the author, pernicious anemia is considered a disease of middle age. His own observations have led him to believe that this is not the case, as during the past five years he has observed fifteen cases of pernicious anemia in patients whose age varied from 65 to 78 years.

Curschmann contends that many cases are overlooked, especially in the aged, and the symptoms complained of attributed to carcinoma, senile marasmus or cardiac insufficiency. The disease would be more frequently recognized if aged persons complaining of sore tongue were examined for pernicious anemia.

The symptoms complained of in the aged are practically identical to those occurring in patients of middle life. The spinal cord symptoms, such as changes in reflexes, paresthesias, anesthesias, alexia and other symptoms are encountered just as frequently.

The author discusses the subject of aplastic anemia and the possibility of atrophic changes in the bone marrow or other physiologic senile changes being the cause of the changes noted. Hereditary constitutional degenerative changes may also play a part.

Three of Curschmann's cases came to necropsy, but no mention is made of microscopic examination of the central nervous system.

In conclusion the author states: Pernicious anemia is not uncommon in the aged. Frequently the disease is concealed under the mask of senile marasmus and cardiac insufficiency. One should make a blood study of every case of glossodynia occurring in the aged. It is not unusual, as in the middle aged, to have long remissions following the energetic use of arsenic.

MOERSCH, Rochester, Minn.

THE PROBLEM OF THE PSYCHOPATHIC CHILD. HENRY H. GODDARD, Am. J. Insan. **77**:511 (April) 1921.

Goddard attempts to formulate certain distinguishing criteria between mental deficiency and juvenile psychopathy. The more important on the basis of the psychametric measurement (Binet-Stanford) and association test (Kent-Rosanoff) are as follows: (*a*) the psychopathic child, while often showing satisfactory comprehension and reasoning ability, is apt to fail in association, weight discrimination, and memory power while the feebleminded child frequently has a rote retentiveness; (*b*) the responses of the psychopath to the Binet questions reveal a prominence of "scatter"; (*c*) the quality of the psychopath's replies are unusual with much individuality and interpellation; (*d*) the psychopath's associations include more than ten individual or more than forty-five common reactions and at least ten abnormal associations.

The further differential diagnosis rests on behavior indications. The points the author makes are excellent, but cooperative study between the psychologist and clinical psychiatrist is needed to decide this phase of the question. Nothing is said of the borderline case. In practice and particularly in outpatient neuropsychiatric work, one is not infrequently confronted with the problem of differentiating from the content between a mere psychotic episode or trend and a beginning psychosis, or again between primary mental deficiency and secondary ill-defined dementia. The decision is especially difficult in the absence of a reliable anamnesis. The presence of "scattering" in the psychometric measurement has considerable value. It may be that Goddard's additional determinations will be even more helpful.

STRECKER, Philadelphia.

EARLY APPEARANCE OF SPINAL CORD SYMPTOMS IN PERNICIOUS ANEMIA (BIERMER). ERICH BALLMANN, Med. Klin. **17**: 249 (Feb. 27) 1921.

The early occurrence of cord changes in pernicious anemia is only superficially touched on in most textbooks. Naegeli, in his latest edition on "Bluterkrankungen und Blutdiagnostik," calls attention to the early appearance of cord symptoms, but is inclined to believe that the blood study if made earlier would have shown signs of the disease.

Ballmann reports four cases in which the cord symptoms were the first indication of trouble; the blood study was made later and the characteristic blood picture discovered. In all cases, paresthesias, gait disturbance and other symptoms long preceded the anemia, and only after careful study was the disease process recognized.

Ballmann is of the opinion that patients showing cord changes are difficult to treat and do not respond as well as those showing little or no cord changes, but a more profound anemia. The four cases reported by the author came to necropsy, and the diagnosis was substantiated.

The author is not certain in what proportion of cases of pernicious anemia cord symptoms are present, but from his personal observations he is inclined to believe that cord changes are more often early than late manifestations of pernicious anemia.

The author brings up the question of fatigue and malnutrition subsequent to the war in relation to cord changes, but draws no conclusions. He finally suggests a careful study of the blood in all system diseases of the cord in which the diagnosis does not seem clear.

MOERSCH, Rochester, Minn.

THE COMPENSATORY MECHANISM OF DELUSIONS AND HALLUCINATIONS. S. A. Silk, Am. J. Insan. **77**:523 (April) 1921.

This article contains the report of two cases from St. Elizabeth's Hospital and a preliminary and general discussion of the biologic demands in man. The first case is the more interesting. A man, aged 29, was admitted to the hospital suffering from delusions of persecution and reference, "somewhat excessive and circumstantial in speech, and melodramatic in his actions." As a boy he was called a "sissy." Since the age of 12 he had had depressions about once a year. Although well educated, he had failed in several occupations; he then enlisted in the army, where he ran up against a very masculine company commander. After six months' residence, he was discharged as a case of social recovery without insight. The older descriptive psychiatry might say that his life was ruined by manic-depressive attacks; whenever he made a start in business a depression threw him back; he varied between brightness and dulness. The newer view taken by the author regards him as a sickly, spoiled child with a strong mother attachment, homosexual and so insecure, trying to show himself and others that he is not weak by joining the army and building up ideas of persecution about his commanding officer.

Bond, Philadelphia.

VARIATIONS IN INTENSITY OF AFFECTIVE REACTIONS. T. Graham Brown, L'Encéphale **16**:201 (April) 1921.

Brown gives an account of some experiments dealing with the effect on respiration of stimulation of the midbrain of decerebrate monkeys. The point which he stimulated lay on the transverse section of the midbrain near the aqueduct and at the level of the superior limit of the anterior quadrigeminate bodies. He shows that with a mild current respiration was slowed. As the current was increased, respiration became more and more rapid, until, with a strong current, there were associated movements of the tail, extremities, trunk and neck. He emphasizes the dependence of the reaction on the variation of the strength of the current. He characterizes the respiratory action as an affective one and offers his result as an argument against the statement of Rivers that an "all or nothing" reaction is one of the characteristics of instinct. He ends by saying, "We therefore are brought to the conclusion that an affective reaction may be of varying intensity and that it is not 'all or nothing.'" This work is another link in the chain of evidence indicating that the visceral nervous system is centrally represented by a third neuron, i. e., one central to both the preganglionic and postganglionic portions of the arc. This communication of Brown's is a continuation of work previously published in the *Journal of Physiology*, 1915 and 1916.

Kraus, New York.

AN EXPERIMENTAL STUDY OF THE SACRAL SYMPATHETIC TRUNK OF THE CAT, WITH SPECIAL REFERENCE TO THE OCCURRENCE OF INTRINSIC COMMISSURAL NEURONS. Sydney E. Johnson, J. Comp. Neurol. **33**:85-104 (April 15) 1921.

The experiments here described yield accurate information regarding the structure and connections of the sacral sympathetic trunk of the cat, the conclusions from which are clearly presented in a diagram. The normal and degenerated tissues were stained with osmic acid and Ranson's pyridin-silver

technic, thus revealing the relations of both myelinated and unmyelinated fibers. The sacral and coccygeal ganglions possess a rich intercellular plexus which completely disappears following descending degeneration of preganglionic axons in the lower lumbar and sacral trunk. It must, therefore, be formed by the terminations of preganglionic efferent axons which run to the trunk through the lower white rami. No evidence was found suggesting the presence of intrinsic commissural or connecting neurons within the sympathetic ganglions themselves or connecting adjacent ganglions. All nervous elements stained by the methods employed were traced to some other source or otherwise accounted for.

C. J. Herrick, Chicago.

TERTIARY SYPHILITIC PSYCHOSES OTHER THAN PARESIS. Burton Peter Thom, Am. J. Insan. **78**:503 (April) 1921.

The text of Thom's article is that "there is no psychosis which cannot be caused by syphilis." Manic-depressive insanity, involutional melancholia and paranoia are drawn in direct relationship with syphilis. One may readily concede an indirect connection, but the intermediate steps are still uncertain. More and more in psychiatry and indeed in internal medicine, must we subscribe to the doctrine of multiplicity of causes. Even in such well-established conditions as paresis, alcohol or toxic psychosis, we are evidently dealing with syphilis and an extraneous poison respectively plus an unknown factor which preciptates the psychosis. This unknown factor deserves the closest investigation and analysis.

Strecker, Philadelphia.

NERVE AND PLASMODESMA. H. V. Neal, J. Comp. Neurol. **33**:65-75 (April 15) 1921.

The problems relating to the histogenesis of the nervous elements have by no means been fully solved by the demonstration, now generally accepted, that nerve fibers arise as outgrowths of embryonic nerve cells. By many it is claimed that these axonic outgrowths pass into a preexisting protoplasmic network of some sort, a plasmodesma. In favorable material of dogfish embryos stained by the silver-reduction method, Neal has seen neurofibrillae in the primary connections between the neural tube and the myotome, and from his observations he concludes that there are no indifferent plasmodesmas which are utilized as pathways by the growing neuraxons. The earliest structures to appear connecting the neural tube and peripheral end organs are true nerve fibers, axons of the embryonic nerve cells.

C. J. Herrick, Chicago.

REMARKS ON UNDIAGNOSED CASES, CHICAGO STATE HOSPITAL, 1919. Charles F. Read, Am. J. Insan. **77**:493 (April) 1921.

The more a hospital staff studies its cases the more it leaves unclassified. Read considers the cases of 146 patients under 60 years of age, and it may well be questioned whether he could not have added many beyond that age. His work emphasizes again that the prognosis is better for the unclassified patients than for all admissions. He concludes that confusion and apprehension in an acute psychosis indicate dementia praecox.

Bond, Philadelphia.

ARE THE TASTE-BUDS OF ELASMOBRANCHES ENDODERMAL IN ORIGIN? MARGARET H. COOK and H. V. NEAL, J. Comp. Neurol. **33**: 45-63 (April 15) 1921.

The specificity of the embryonic germ layers is again attacked in this contribution, in which the authors present evidence that both taste-buds and pharyngeal scales arise in the dogfish from entoderm. These pharyngeal scales resemble in structure the placoid scales of the skin, which are undoubtedly of ectodermal origin. In some other fishes (catfish, carp) taste-buds essentially similar to those within the mouth and pharynx are found freely distributed in the outer skin of the head and trunk, and these have been shown by Landacre to be of ectodermal origin. These observations tend to show that adult structures of similar type may develop from either ectoderm or entoderm, as occasion may arise.

C. J. HERRICK, Chicago.

THE FIRST THORACIC WHITE RAMUS COMMUNICANS IN MAN. S. E. JOHNSON and M. L. MASON, J. Comp. Neurol. **33**:77-84 (April 15) 1921.

Existing descriptions of the white ramus communicans related with the first thoracic spinal nerve are vague and contradictory. Examination of twelve laboratory bodies and of fresh necropsy material showed in each case one or more white rami arising from the first thoracic nerve and connecting, as a rule, with the stellate ganglion. These rami contain a relatively high percentage of large myelinated fibers, whose distribution offers a further problem of some interest. Variations in the arrangement of these rami are described and figured.

C. J. HERRICK, Chicago.

TREATMENT OF MELANCHOLIA BY STRYCHNIN IN VERY HIGH DOSAGE. P. HARTENBERG, Progrès méd. **36**:123 (March 19) 1921.

Hartenberg points out that melancholic depression is a disturbance of affectivity and of cenesthesia and believes that strychnin in large dosage, by exerting a powerful influence on the vegetative nervous centers, is beneficial. In a series of six cases, he has obtained a cure in five cases and improvement in one.

Using a 1 per cent. solution of the sulphate of strychnin, he commences by giving the first day 7 drops (0.0035 gm. dosage), three times daily, either by mouth or injection. Then, each following day, he increases each dose by one drop. If at the end of several days dizziness and stiffness of the legs and jaws appear, the dose is held stationary until the reaction disappears. Then he again augments the dose as before. One then comes to the point where the same dose always provokes a reaction. This marks the phase of saturation and the dose cannot be further increased. It requires a month approximately to reach the dose (0.07-0.08 gm.) which marks this saturation.

"The progress of the cure parallels the medication." When a dosage of 0.05 gm. is passed, the patient begins to wake, to talk, to occupy himself and to smile.

The writer considers that it is only because strychnin has always been used in too small dosage that its therapeutic value in psychotic depressions has never been recognized

DAVIS, New York.

FEEBLEMINDEDNESS IN HEREDITARY NEUROSYPHILIS. O. J. RAEDER, Am. J. Dis. Child. **21**:3 (March) 1921.

This paper deals with the incidence of neurosyphilis and feeblemindedness in children of syphilitic parents, and quotes an incidence of 38 per cent. reported by Jeans, and an estimate of 33 per cent. reported by other authors. The author collected thirty cases, the children ranging in age from 2 to 16, whose parents one or both, were syphilitic, most of them neurosyphilitic and usually paretic. In the spinal fluids from twenty-two of the patients, four were positive to the Wassermann reaction and nineteen had a positive blood reaction. The children born nearest the time of the parental infection gave positive reactions while the youngest in a family gave negative reactions. Of the four patients with a positive spinal fluid, the gold curve fell within the "paretic zone." As to the mental condition of these children, those farthest away from the parental infection approached nearest to normal, but it is observed, that though seronegative, they were somewhat backward mentally. The greatest mental defects were found in the older children. In one family in which the father was paretic and the mother became so later on, there were five children ranging in age as follows: 13, 12, 9, 8 and 4. Their mental ages were respectively: $6\frac{8}{12}$, $8\frac{3}{12}$, $7\frac{4}{12}$, $4\frac{10}{12}$ and $3\frac{8}{12}$. The author holds to the view that the syphilitic toxin must have been the cause of the mental defect as the parents were definitely not feebleminded. In another family of four children, the first, who was born before the parental infection, was healthy, the second had a psychosis with all tests positive for neurosyphilis, the third had a positive blood Wassermann, while the fourth was entirely negative. Other children of the series showed a mental age below the actual age, and among them were truants, backward scholars and one with hydrocephalus. One patient only showed a mental age above the actual age.

PATTEN, Philadelphia.

CEREBRAL SOFTENING DUE TO VENOUS THROMBOSIS IN PUERPERAL INFECTION. R. CREMIEU and P. GAUTHIER, Progrès méd. **36**: 101 (March 5) 1921.

This paper includes the reports of two cases of hemiplegia in the puerperium which developed in persons with postpartem infection and pelvic phlebitis. Hemiplegia developed with great deliberateness, in the first case requiring three days. It was possible to exclude syphilitic, eclampsic and nephritic factors as possible etiology. In neither case was there an endocarditis.

The writers believe that whenever a cerebral softening manifests itself in the course of puerperal infection, when the integrity of the heart permits one to eliminate the hypothesis of an embolus, a venous thrombosis is indicated rather than an arterial thrombosis. This diagnosis is rendered plausible by necropsy examinations which can be found reported in the literature, and is strengthened by the known special affinity of the puerperal streptococcus for the venous system.

The patients in the two cases reported both recovered. No opinion is expressed concerning the probable prognosis in most cases.

DAVIS, New York.

PARALYSIS OF THE LEFT RECURRENT LARYNGEAL NERVE ASSOCIATED WITH MITRAL STENOSIS. J. GARLAND and P. D. WHITE, Arch. Int. Med. **26**:343 (Sept.) 1920.

In a review of the literature the authors found sixty-one cases of this condition reported. The theories as to the anatomico-etiologic factors are discussed briefly. It is claimed by some writers that enlargement of the left auricle causes pressure atrophy or neuritis of the recurrent laryngeal nerve. Others state that the pressure must be exerted between the pulmonary artery and the aorta or aortic ligament. One writer states that elongation of the nerve is produced by dilatation of the right ventricle. Mediastinitis and thrombosis in the left auricle are given as causes. The authors agree that the pulmonary artery is an intermediate agent in causing pressure, and that occasionally a chronic mediastinitis or a thrombosis in the left auricle are etiologic factors. Auricular fibrillation—by keeping the auricle balooned up, readily producing thrombi, is an added factor.

Nine cases are reported briefly. Summing up the most frequent findings, it is shown that the roentgen ray revealed that three patients had an enlargement of the left auricle. Two patients had enlarged right pupils. Auricular hypertrophy and right ventricular preponderance were present in three, while ventricular preponderance alone was present in two. Four patients had auricular fibrillation. The electrocardiogram of one patient revealed a cardiac displacement from an extracardiac cause. There was possible adherent pericarditis in addition to the mitral lesions in three of the patients.

Hoarseness, cough and dyspnea were present sooner or later in every case, but the duration of hoarseness varied in time before admission from five days to ten months. The authors believe the condition is frequently overlooked.

PATTEN, Philadelphia.

THE COLLOIDAL GOLD CURVE IN EPIDEMIC ENCEPHALITIS. A PRELIMINARY NOTE. T. K. DAVIS and WALTER M. KRAUS, Am. J. M. Sc. **161**:585 (Jan.) 1921.

A table is given showing the colloidal gold reaction in thirty-four cases of epidemic encephalitis. A study of this table shows that in twenty-five fluids the reaction was definitely negative, giving a zero curve, while in the other nineteen fluids a curve was obtained ranging from 5544332100 to 0000110000. The number of cases showing changes totals 41 per cent. It is noted that the higher curves suggest syphilis, but the serologic studies of the blood and spinal fluid were negative in this respect. The cell counts in the fluids show no parallelism with the colloidal gold curve, ranging from 0 to 134. In some cases three specimens were examined from the same patient, but there were no constant findings. In considering the duration of the disease, the cells seem to be most increased in the third week, but the colloidal gold reaction was less marked at this time than during the seventh to the twelfth weeks. The author concludes that there is no relation between the gold curve and the severity, duration or etiology of the disease. In a fairly large percentage of cases, however, changes of some sort are shown, which indicate a pathologic condition.

PATTEN, Philadelphia.

THE SPINAL FLUID IN EPIDEMIC ENCEPHALITIS. J. A. BARRÉ and L. REYS, Bull. méd. **35**:366 (April 27 and 30) 1921.

These writers report that in their cases of epidemic encephalitis, chemical and cytologic analysis of the spinal fluid showed hypercytosis in 32 cases, hyperglycosis in 32, hyperalbuminosis in 24 and normal fluid in 19. The maximum cell count was 105. The maximum albumin content 30 mg., and the maximum sugar content 110 mg. per 100 c.c. Increase in sugar alone occurred in 15 cases, of albumin, sugar and cells in 11 cases, of albumin and cells in 9, of cells alone in 8 cases, of cells and sugar in 4 cases, of albumin and sugar in 3 cases, of albumin alone in 1 case.

Thus the meningitis of epidemic encephalitis, which evidences itself clinically infrequently, causes definite modifications of the spinal fluid. The augmentation of the sugar content, however, is the characteristic feature of the meningitic reaction.

Its evolution has no parallelism with oscillations in the clinical course.

The spinal fluid remains abnormal for three, four or even eight months, and for this reason the writers never cease treatment in a case or allow the patient to resume a usual regimen until lumbar puncture proves that the spinal fluid has returned to normal.

DAVIS, New York.

DISCRETE THERMAL CHANGES IN SPINAL SYPHILIS. CHARLES A. MCKENDREE, Neurol. Bull. **3**:144 (April) 1921.

McKendree describes a case of spastic paraplegia in a man of 38, probably of syphilitic origin, which came on gradually and was accompanied by urinary disturbance since the onset, three years before. The cord changes were apparently limited to the pyramidal tracts, there being no sensory changes except gross disturbance or loss of appreciation of hot and warm stimuli in both lower extremities. Cold was perceived throughout, and pain sensation was preserved. This unusual dissociation in the appreciation of cold and warm stimuli and pain sensation speaks for a theoretical dissociation of the fibers in the spinal cord, or at least the possibility of their being so discrete that they can be individually damaged. It is rather difficult to explain this dissociation in the spinothalamics which are presumed to carry the pain, heat and cold fibers. The reviewer has seen a similar case in which perception of pain and cold was preserved while that of heat was lost. Further clinical confirmation supported by pathologic findings may lead to more exact knowledge of the sensory pathways in the cord.

WECHSLER, New York.

WEICHBRODT'S SUBLIMAT REACTION IN THE CEREBROSPINAL FLUID. EMIL JOSEPH MARX, Monatschr. f. Psychiat. u. Neurol. **48**:227 (Nov.) 1920.

In 1916, Weichbrodt reported his sublimat tests (seven parts of spinal fluid and three parts $\frac{1}{1000}$ solution of mercury bichlorid). He asserted that a positive reaction (opalescence, turbidity) meant syphilis. Later he found that certain organic, nonsyphilitic diseases of the brain produced a similar reaction. He found the two reactions different, however—those due to syphilis giving a bluish cast.

Control of these results by other workers has not borne out Weichbrodt's assertion. Marx examined 107 fluids from syphilitic and nonsyphilitic patients, with special reference to the sublimat test. He found it of no especial value. It has approximately the same significance as the Nonne test—phase 1.

SELLING, Portland, Ore.

(A) VON RECKLINGHAUSEN'S DISEASE IN THE NEGRO; (B) CURVATURE OF THE SPINE IN VON RECKLINGHAUSEN'S DISEASE. R. S. WEISS, Arch. Dermat. & Syph. **3**:44 (Feb.) 1921.

(a) This is a report of two cases of this interesting disease in the negro. Although occurring in the colored race it does not essentially differ from that seen in white people.

(b) Of greater interest is the report of curvature of the spine associated with this disease in all cases observed at the clinic. There was likewise found in all cases a definite abnormality of the mentality, and in women menstrual disturbance was frequent, which leads the author to suggest the possibility of endocrinous defects as an etiologic factor.

PATTEN, Philadelphia.

DISAPPOINTMENTS OF ENDOCRINOLOGY. EDITORIAL, J. A. M. A.

A recent issue of *The Journal*[1] contains a caution lest, in the current enthusiasm for so-called endocrinology, medicine become humiliated by the drift toward a sort of pseudoscience bolstered up with meaningless words and unfounded assumptions. It is time for the medical profession to face the facts squarely. We shall not deny the great advances that have been made in comparatively few years in the study of thyroid function. Much of the knowledge gained either by scientific research or by clinical observation has "made good" through direct application in practical therapy. Changes in thyroid function may lead to symptoms that are fairly definite, determinable, clinical entities. Therapeutic success attests the value of much that has been accomplished and the verity of a few of the assertions that are taught.

As much cannot be said for the suprarenal structures. G. N. Stewart of the Western Reserve Medical School at Cleveland deserves the thanks of the medical profession for the fearless and critical manner in which he has questioned much of the verbal rubbish that goes under the designation of the endocrinology of the suprarenals. In a trenchant review he[2] has gone even farther in pointing out the invalidity of some of the experimental evidence that has been marshaled to support pet theories. One postulate stands unchallenged, namely, that the loss of the suprarenals is incompatible with life. Hence the conclusion is inevitable that these structures contribute something indispensable to the functioning of the body.

What, then, is "suprarenal insufficiency?" To this question no one has yet given a satisfactory answer. The symptoms attending decline and death after extirpation of the suprarenals are not specific or constant; indeed, they are

1. The Endocrine Glands.—A Caution, editorial, J. A. M. A. **76**:1500 (May 28) 1921.

2. Stewart, G. N.: Adrenal Insufficiency, Endocrinology **5**:283 (May) 1921.

duplicated by the outcome of various other abnormal states in which no "endocrine" disorder is assumed. The essential manifestations of the loss of the suprarenals remain obscure. Why shall we continue to postulate a condition which cannot be diagnosed even under the most favorable circumstances?

The first answer to any query about the suprarenals is likely to involve the function of epinephrin, the well defined chemical compound that is usually present in the medullary portions and can somehow be discharged into the circulation. The pharmacologic potency of this product cannot be denied. It was natural that attention should be directed promptly to the possibility that epinephrin is the primary active principle, the physiologic hormone of the suprarenals. Is "suprarenal insufficiency" represented, then, by interference with the output of epinephrin? This can scarcely be the case; for animals continue in health after extirpation of one suprarenal and denervation of the other, a procedure after which no detectable amount of epinephrin is liberated. The assertion that epinephrin is at least secreted in physiologic emergencies has been reviewed in *The Journal*[3] with the conclusion of "not proved." The assumption that it serves to maintain the tonus of the blood vessels and the normal blood pressure has likewise proved to be untenable. Injected epinephrin has a definite pharmacologic action on the circulation; so have drugs not even remotely associated with animal tissues. Neither class necessarily represents a normal stimulus because it happens to have demonstrable effects when introduced in the body. No one alleges that the alkaloids, powerful though they are, have a known physiologic function in plants.

Furthermore, although epinephrin is a component of the suprarenal medulla, it is the cortex of the suprarenal structures that appears to be the vitally important portion. If one argues for the "fetish of suprarenal insufficiency," it can scarcely be associated with the hormone epinephrin; for, as Stewart points out, the experimental evidence has tended more and more to show that the cortex is the part of the suprarenal indispensable for life. It is the cortical tissue which alone, or at any rate most conspicuously, undergoes compensatory hyperplasia when a deficiency is created by removal of a considerable part of the suprarenal tissue. The accessory suprarenal tissue, which also undergoes hypertrophy under these circumstances, and which is usually supposed to be responsible for the survival of those animals that do not succumb to the loss of both suprarenals, consists entirely of cortex. Only the cortical tissues can be successfully grafted.

Even in Addison's disease there is no experimental basis for the assumption that the defects of suprarenal function are due to a loss of epinephrin function. How much, then, can be implied in the vague "hypoadrenal" states of clinical literature? There is something stinging, yet deserved in its implied rebuke, in the words of Stewart.[3] "On the whole," he says, "it must be granted that hitherto the attempts made to evoke in animals a well marked syndrome characteristic of adrenal deficiency have been singularly disappointing. The contrast is great when we leave this desert, where the physiologists and experimental

3. Is Epinephrin Indispensable to Life? editorial, J. A. M. A. **73**:192 (July 19) 1919; The Debated Theories of Suprarenal Function, ibid. **74**:326 (Jan. 31) 1920.

pathologists have wandered, striking many rocks but finding few springs, and pass into the exuberant land of clinical endocrinology, flowing with blandest milk and honey almost suspiciously sweet."

How much longer will our profession continue to merit such criticism? Just so long as our profession continues to give serious consideration to pseudo-scientific rubbish promulgated by the exploiters of organic extracts.

Book Reviews

THE LOGIC OF THE UNCONSCIOUS MIND. By M. K. Bradby. Oxford Medical Publications, London: Oxford University Press, 1920. Pp. 316.

In his introduction to this work the author expresses his thought that the science of logic has to be reformed because we have "outgrown the logic of our forefathers just as we have outgrown their chemistry—a fact which leading logicians are quick to recognize." It occurs to the reviewer that this is hardly a correct statement of the situation. Logicians and students of logic, along with the rest of the population, may always be expected to catch the changing spirit of the times. Just now these logicians and students recognize a demand that logic, along with other disciplines, is required to relate itself to daily life rather than to dead issues. The reforming of logic, to which we are called, is only a new adaptation of the science. The author goes on in his introduction to say that the student of logic is called on to give the subject a fresh start "and this he is enabled to do by the discoveries of psycho-analysts concerning the unconscious mind"—in other words, he might have said that a particular branch of science has brought to light within recent years new data which the logician is called on to reckon with.

The volume under review is taken up with a discussion of these new data which comprise a great body of detail, more or less well verified, that have been brought to light by the psycho-analysts and psychologists of the last several years. Altogether, these data comprise the unconscious background of conscious reasoning which the author discusses in Part 1 of his volume under the titles "Instinct," "Intuition," "Dreams and Unconscious Symbolism," "Language," and "Some Logical Implications of Language."

In all this unconscious background of conscious reasoning the author finds the unconscious motives which are the source of fallacy. These motives he discusses in Part 2 in four chapters entitled, respectively, "Fallacies of Observation and of Conduct," "Some Typical Cases of Fallacy Analyzed," "Fallacies Classified" (two chapters). They are our predisposition, prejudices, etc. Finally, in Part 3 he shows how logic, rewritten in the light of the data supplied by an analysis of the unconscious background, is applied to life; and this he discusses in several chapters entitled, respectively, "Popular Misconceptions Concerning Reason, Logic and Education," "The Logic of Compromise, Logic and Social Problems," "Logic and the Phenomena of Spiritualism, Logic, Love and Religion," "The Logic of God and Devil," "Conclusions," "Progress."

It seems to the reviewer that the present volume is not so much a treatise of logic as it is a discussion of a certain aspect of psychology—that aspect of the new psychology that laps over on the data of the psycho-analyst. This new psychology finds in the data referred to a root of human conduct whether that conduct be the formation of language, the writing of a fable, a dream, the composition of a poem, the attitude toward social problems such as education, or what not. The author has discovered no new logic and no new logical method. He has only pointed out some natural facts, hitherto but partially recognized, that logic must deal with and some pitfalls that it must avoid.

DER EXTRAPYRAMIDALE SYMPTOMENKOMPLEX (DAS DYSTONISCHE SYNDROM) UND SEINE BEDEUTUNG IN DER NEUROLOGIE. Von Prof. Dr. G. Stertz, Oberarzt der Psychiatrischen Klinik in München. Price, 18 marks. Pp. 96. Berlin: S. Karger.

This monograph gives an excellent résumé of the various symptom groups due to disease of the extrapyramidal motor system or systems with some interesting and instructive personal cases. After a brief historical introduction, Wilson's disease and pseudosclerosis are described and compared, the conclusion being that they are essentially the same disease. Then paralysis agitans in its different types and variations is considered, and while the author believes that the anatomic basis of this disease has not been determined, he believes that there is such a basis and that the disease is closely related to Wilson's disease and pseudosclerosis.

The author then takes up in turn the "akinetic-hypertonic" syndrome, double athetosis, torsion spasm (dystonia musculorum deformans), the spastic-athetotic syndrome, the choreic syndrome and myoclonus.

Part 2 covers the application of the knowledge gained by a study of these different extrapyramidal motor disorders to neurologic diagnosis. That is, he shows how a hemorrhage, vascular occlusion, tremor, multiple sclerosis or encephalitis, located in the striatum region, may produce a syndrome very much like paralysis agitans, Wilson's disease, etc. Finally, there is a pathophysiologic summing up of the entire situation with the suggestion that this entire group of diseases be classed together under the designation "dystonic syndrome."

While the author does not claim to cover the literature, his bibliographic omissions outside of Germany are notable. For instance, the papers by Ramsay Hunt are not mentioned, there is only one French reference and Wilson is the only English author quoted—and he is quoted rather grudgingly.

FEEBLENESS OF GROWTH AND CONGENITAL DWARFISM. WITH SPECIAL REFERENCE TO DYSOSTOSIS CLEIDO-CRANIALIS. By Dr. Murk Jansen, O.B.E., Lecturer on Orthopedic Surgery, University of Leiden, Holland. Pp. 82. Oxford Medical Publications. London: Henry Frowde and Hodder and Stoughton, 1921.

This is a further elaboration of principles laid down in previous contributions by the author, particularly in his monograph (1912) on achondroplasia. Part 1 deals with "feebleness of growth" and is a defense of the following three theses: "1. Feebleness of growth is proportional to the intensity of the nocivity. 2. Feebleness of growth is proportional to the rapidity of the growth of the individual and its parts (law of the vulnerability of fast-growing cell groups). 3. Feebleness of growth is characterized by enhanced sensibility and enhanced fatigability."

Part 2 is devoted to congenital dwarfism, with a lengthy and profusely illustrated discussion of dysostosis cleido-cranialis and brief chapters on anencephaly, achondroplasia and mongolian idiocy. All of these conditions are considered as caused by smallness of the amnion becoming operative at various periods of fetal life.

The philosophic and thoroughly logical, as well as original, treatment of obscure developmental conditions makes the book most interesting and stimulating. However, the purely mechanical factors appear somewhat overem-

phasized, while surprisingly little attention is given to endocrine factors, the importance of which, in disorders of growth, is becoming more generally recognized by other workers in this field.

LEITFADEN DER NEUROLOGISCHEN DIAGNOSTIK. Eine Differentialdiagnose aus dem führenden Symptom für praktische Aerzte und Studierende. Von Dr. Kurt Singer, Nervenarzt in Berlin Neurologischer Fachbeirat beim Hauptversorgungsamt. Paper. Price, 45 marks. Pp. 201, with 33 illustrations. Berlin: Urban & Schwarzenberg, 1921.

The conscience of the author of this little compendium seems to have been a bit uneasy as he takes about four pages to justify his product. Without greatly abusing hyperbole, one might call it a childish book for children. On that basis it may be considered quite useful although it contains little not to be found in any good textbook.

There are eighteen chapters, each devoted to a principal group of symptoms of disease or disorder of the nervous system: paralyses, disturbances of sensation, muscular atrophy, disorders of gait, spasms, tremor, disorders of speech, etc. In general, the diagnostic factors are good, but one receives the impression that the author is neither experienced nor careful. Even a cursory reading reveals errors. For instance, the statement is made (page 17) that a cord lesion below the cervical enlargement, where the nerves for the arms have already been given off but not those for legs, causes a flaccid paralysis of the arms and spastic paralysis of the legs. Facial spasm is (page 80) inextricably mixed with facial tic, and at one place is called impulsive tic, whereas it is neither impulsive nor a tic. The diagnostic criteria of facial neuralgia are pitifully inadequate, the author depending almost entirely on the Valleix points which, as a matter of fact, are generally absent and never of value.

In short, this little book may be helpful to the student and general practitioner (for whom so many sins are committed) but not so helpful as a bigger and better book.

HOW THE MIND CURES A Consideration of the Relationship Between Your Outside and Your Inside Individualities and the Influence They Exercise on Each Other for Your Physical and Mental Welfare. By George F. Butler, A.M., M.D., Medical Director, North Shore Health Resort, Winnetka, Ill.; formerly Professor of Materia Medica, Therapeutics and Clinical Medicine, College of Physicians and Surgeons and Chicago College of Medicine and Surgery. Price, $2.50. Pp. 286. New York: Alfred A. Knapp, 1921.

In the publisher's prospectus it is claimed that "this book tells how the mind cures and in so simple and interesting a manner that anyone who reads it will find that which will teach him how to be and keep well, filling him with delight and giving him a sense of increased power and poise." This is a large order, but it must be admitted that the author has attacked his subject in a sane and conscientious manner and in entertaining form, as a dialogue between the physician and the seeker for all of these benefits. The book gives sensible answers to a great many questions constantly asked by neurotic patients of the educated class and may safely be placed in their hands.